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RESPIRATORY CARE

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SPECIAL ISSUE
Controversies in Home Respiratory Care
Part I

- Controversies in Home Respiratory Care: A Conference Summary
- Demographics and Financial Impact
- Current and Future Role of RCPs
- Controversial Indications for Long-Term Oxygen Therapy
- Travel for the Technology-Dependent Individual
- Reservoir Cannula, Demand Flow & Transtracheal Oxygen Delivery
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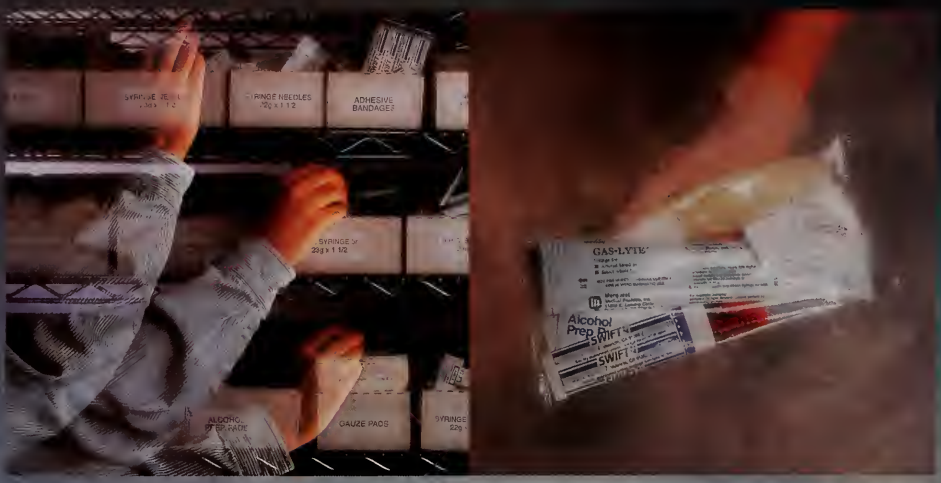

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Disposition and Respiratory Effects of Intrathecal Morphine in Children (highlights)—DR Stanski. Anesthesiology 1993;79(4):25A. (Pertains to Nichols et al paper abstracted on Page 284.)

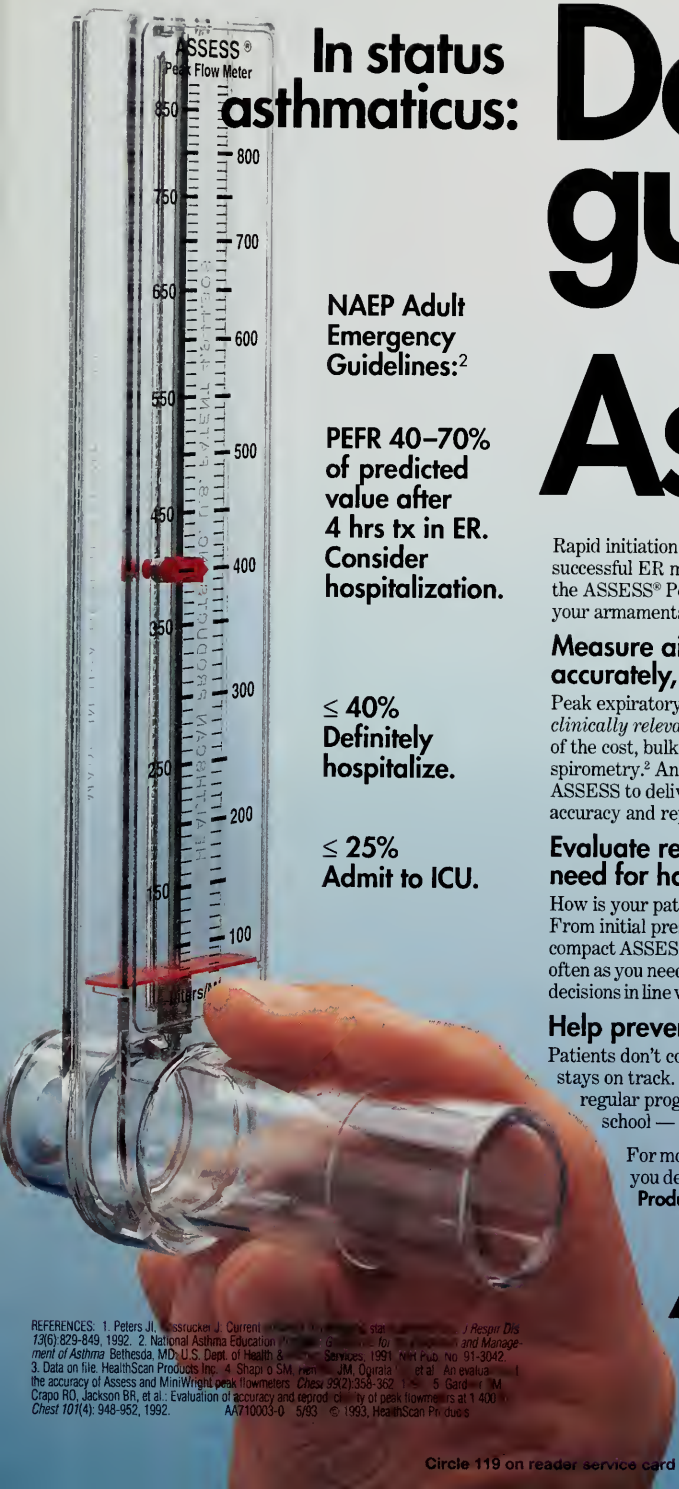
Delivery Dependent Oxygen Consumption: Asking the Wrong Questions and Not Getting Any Answers (editorial)—M Smithies, DJ Bihari. Crit Care Med 1993;21(11): 1622. (Pertains to De Backer et al paper abstracted on Page 289.)

Severe COPD and Acute Respiratory Failure: Correlates for Survival at the Time of Tracheal Intubation—RD Rieves, D Bass, RR Carter, JE Griffith, JR Norman. Chest 1993;104:854.

The recognition of a reversible cause for acute respiratory failure (ARF) is frequently difficult in patients with severe chronic obstructive pulmonary disease (COPD). We sought to identify clinical findings present at the time of tracheal intubation that were associated with successful weaning and short-term survival among a population of male veterans with severe COPD. Over a 5-year period (1987 to 1991), 39 episodes of ARF requiring mechanical ventilation (MV) were identified in 33 men with severe COPD. All the patients had a baseline $FEV_1 < 1$ L. Univariate analysis suggested a higher serum albumin level and absence of pulmonary infiltrates on chest radiography distinguished survivors (weaned from MV for 72 h) from nonsurvivors (died while undergoing MV or within 72 h of weaning). Multivariate analysis revealed the absence of pulmonary infiltrates on initial chest radiography was the strongest correlate for survival. To examine the significance of these correlates in ARF complicating milder COPD, 19 patients with lesser degrees of chronic airways obstruction and ARF were also studied. Unlike patients with severe COPD, the presence or absence of pulmonary infiltrates on chest radiography was not correlated with survival in patients with milder chronic airways obstruction. Analyzing all COPD patients with ARF, the mortality risk associated with the presence of pulmonary infiltrates on chest radiography increased dramatically with declining baseline lung function. Mortality risk ratio analysis revealed the greatest likelihood for survival was predicted by a higher baseline FEV_1 and the absence of pulmonary infiltrates on chest radiography. The extent of baseline airways obstruction alone was not correlated with short-term survival in either group. These observations suggest that in the subset of patients with severe COPD and ARF, the presence of pulmonary infiltrates on chest radiography at the time of tracheal intubation may be associated with less likelihood for survival. An exacerbation of COPD may infrequently be the terminal illness in these patients.

Predicting Complications after Pulmonary Resection: Preoperative Exercise Testing vs a Multifactorial Cardiopulmonary Risk Index—SK Epstein, LJ Faling, BDT Daly, BR Celli. Chest 1993;104:694.

Recent studies have used preoperative cardiopulmonary exercise testing to improve risk assessment of pulmonary resection for lung cancer. These studies have demonstrated inconsistent correlation between peak oxygen uptake ($\dot{V}_{O_2}$) and postoperative complications but have not systematically examined other methods



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of risk stratification. We analyzed the findings in 42 patients who had cardiopulmonary exercise testing prior to lung cancer resection. Preoperative clinical data combining pulmonary factors (obesity, productive cough, wheezing, tobacco use, ratio of the forced expiratory volume in 1 s over the forced vital capacity [FEV_1/FVC] < 70%, and P_{aCO_2} > 45 torr), and an established cardiac risk index were used to generate a cardiopulmonary risk index (CPRI). When analyzed using the risk index, the incidence of postoperative complications increased with higher CPRI scores. Those with a CPRI of 4 or greater were 22 times more likely to develop a complication, compared to a CPRI of less than 4 ($p < 0.0001$). We found that patients with a peak $\dot{V}_{O_2}$ less than 500 mL/m^2/min (body surface area) were 6 times more likely to experience a cardiopulmonary complication ($p < 0.05$). With multiple logistic regression analysis, peak $\dot{V}_{O_2}$ was not an independent predictor of postoperative complications. Analysis also demonstrated that a CPRI of 4 or greater was associated with significant reductions in peak $\dot{V}_{O_2}$. We conclude that both the peak $\dot{V}_{O_2}$ during cardiopulmonary exercise testing and a multifactorial CPRI are highly predictive of complications after lung resection. Adding the peak $\dot{V}_{O_2}$ did not enhance the risk estimation generated by the CPRI. The association between postoperative complications and peak $\dot{V}_{O_2}$ may be explained by the correlation between identifiable cardiopulmonary disease (CPRI) and reduced oxygen uptake with exercise.

Vitamin A Supplementation and Increased Prevalence of Childhood Diarrhoea and Acute Respiratory Infections—SK Stansfield, M Pierre-Louis, G Lerebours, A Augustin. Lancet 1993;341:578.

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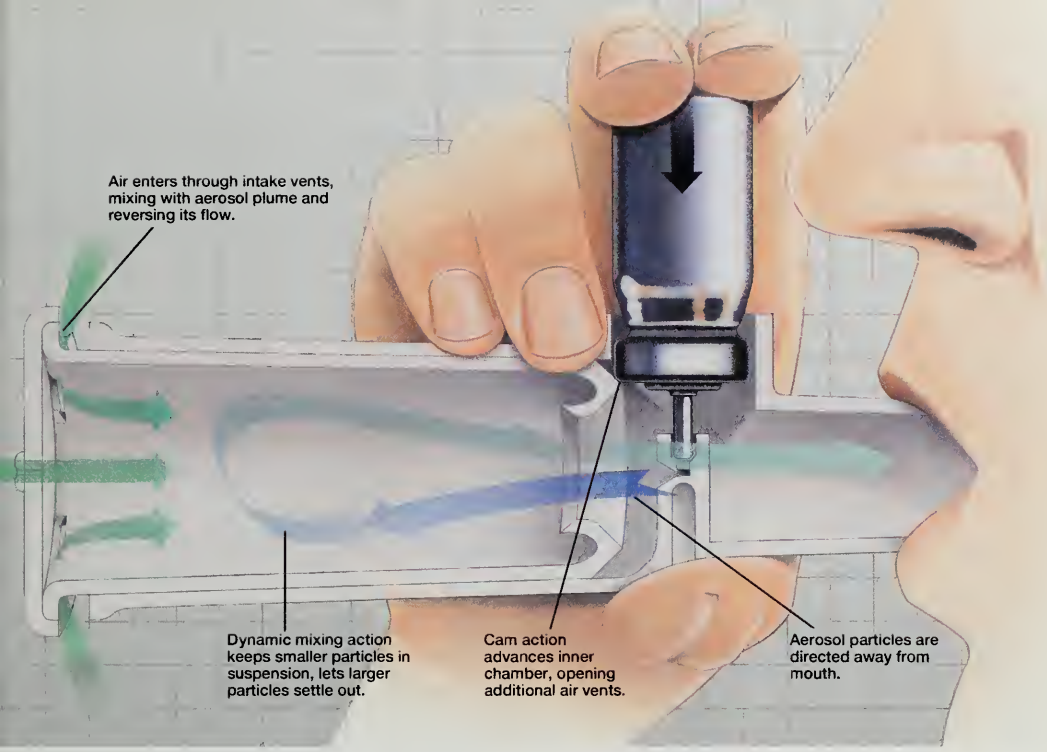
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confidence interval 1.03-1.11), and rapid breathing (RR = 1.18, 95% confidence interval 1.09-1.27). The study shows an increased 2-week prevalence of diarrhoea and the symptoms of respiratory infections after vitamin A supplementation.

Preoperative Evaluation of Cardiac Failure and Ischemia in Elderly Patients by Cardiopulmonary Exercise Testing—P Older, R Smith, P Courtney, R Hone. Chest 1993;104: 701.

Major surgery in the elderly continues to have a high mortality rate. Preoperative myocardial ischemia is a known risk factor. Cardiac failure is also a risk factor, but is difficult to quantify objectively. One hundred eighty-seven elderly surgical patients were evaluated for cardiac failure by cardiopulmonary exercise testing (CPX). The overall mortality in these patients was 7.5%. If three deaths secondary to surgical causes are excluded, mortality was 5.9%. There were 55 patients in whom the anaerobic threshold (AT) was less than 11 mL/min/kg; of these, 10 died, a mortality rate of 18%. There were 132 patients with an AT of greater than 11 mL/min/kg and of these, 1 patient died, giving a mortality rate of 0.8% ($p < 0.001$). A low AT associated with preoperative ischemia resulted in the death of 8 of 19 patients, a mortality rate of 42%. When the ischemia was associated with the higher AT, then 1 patient out of 25 died, a mortality rate of 4% ($p < 0.01$). Both preoperative ischemia and preoperative cardiac failure are independent risk factors for perioperative mortality in the elderly.

Hypoxemic Episodes of Patients in a Postanesthesia Care Unit—GB Russell, JM Graybeal. Chest 1993; 104:899.

The incidence, degree, and duration of acute hypoxemia were evaluated with continuous arterial hemoglobin oxygen saturation monitoring by pulse oximetry in 100 postoperative patients during 40% oxygen administration by aerosol face tent from postanesthetic recovery room admission to discharge. Saturations were recorded by pulse oximeters (Nellcor N 200) with desaturations of $\leq 92\%$ for ≥ 30 s considered significant. On recovery room admission, 15% of patients were experiencing episodes of

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The National Center for Home Mechanical Ventilation has released the "**Learning Objectives for Positive Pressure Ventilation in the Home.**" This 39-page listing of potential educational objectives allows respiratory care professionals to customize patient/caregiver education based upon needs of individual patients. The goal of these objectives is to enhance the education of patients and caregivers in the home. Use of educational objectives will enhance the safety and efficacy of home positive pressure ventilation. The objectives have been developed with the input of numerous healthcare professionals and professional organizations. The objectives are available for a nominal fee.

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desaturation. Low admission saturations correlated positively with patient age and body weight, American Society of Anesthesiologists class, patients having received general anesthesia, and with greater volumes of intraoperative intravenous fluids, particularly > 1,500 mL. Later desaturations to 86.7 ± 4.6% (72 to 91%) at 32 ± 54 min after admission for 5.2 ± 12.6 min occurred in 25% of patients and correlated positively with peripheral surgical procedures, low oxygen saturation on admission, duration of anesthesia, and volume of intraopera-tive intravenous fluids. Desaturation durations were longer for female subjects and correlated positively with body weight and intravenous fluid volume. Significant arterial hemoglobin oxygen desaturations occurred despite prophylactic oxygen administration by aerosol face tent during short-term postoperative recovery room care.

Growth and Pituitary-Adrenal Function in Children with Severe Asthma Treated with Inhaled Budesonide—B Volovitz, J Amir, H Malik, A Kauschansky, I Varsano. N Engl J Med 1993;329:1703.

BACKGROUND: The increased use of inhaled corticosteroids in the management of asthma raises concern about the safety of these drugs in children. We sought to determine the safety of long-term administration of inhaled budesonide in young children with asthma. METHODS: We studied 15 children 2 to 7 years old who had severe perennial asthma. They inhaled 100 μg of budesonide twice daily for 3 to 5 years. Efficacy was assessed by serial evaluation of respiratory symptoms and the need for other medications, and safety by serial evaluation of height, height velocity, weight, bone age, and pituitary-adrenal function. RESULTS: The severity of asthma decreased within the first month after the initiation of therapy, as demonstrated by a 58% reduction in the number of days with symptoms of asthma and a 75% decrease in the use of bronchodilators. This improvement was maintained thereafter. The growth pattern of all patients, including their height, weight, and bone age, was normal (as compared with standard normal values) throughout the treatment period. Pituitary-adrenal function was not adversely affected by the treatment, as demonstrated by normal serum cortisol concentrations in the morning and 60 minutes after stimulation with corticotropin, normal 24-hour serum cortisol concentrations (mean [± SD] of samples collected at 30-minute intervals for 24 hours, 8.4 ± 4.2 μg per deciliter [232 ± 116 nmol per liter]), and normal urinary cortisol excretion (34 ± 9 μg [95 ± 25 nmol] per day). CONCLUSIONS: Prolonged administration of 200 μg of inhaled budesonide daily to young children with severe asthma does not impair growth or pituitary-adrenal function.

Distinct Criteria for Termination of Resuscitation in the Out-of-Hospital Setting—MJ Bonnin, PE Pepe, KT Kimball, PS Clark Jr. JAMA 1993;270(12):1457.

OBJECTIVE: To identify distinct criteria for appropriate on-scene termination of resuscitation efforts for out-of-hospital cardiac arrest when on-scene interventions fail to restore spontaneous circulation. DESIGN: For 18 months, all out-of-hospital cardiac arrests were evaluated prospectively for survival to hospital dis-

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charge and for all established survival predictors including age, gender, presenting cardiac rhythm, whether it was a witnessed event, performance of basic cardiopulmonary resuscitation by bystanders, and interval to paramedic arrival and return of spontaneous circulation (ROSC). SETTING: A large municipality with a single, centralized emergency medical services program. PATIENTS: All normothermic adults treated for out-of-hospital, unmonitored, primary cardiac arrest. INTERVENTIONS: Standard advanced cardiac life support provided at the scene by paramedics. MAIN OUTCOME MEASURES: The number and circumstances of patients achieving survival to hospital discharge following failure to achieve on-scene ROSC. RESULTS: Of 1,461 consecutive primary cardiac arrests, 139 were monitored (paramedic witnessed), including 59 that occurred en route to the hospital. Of the 1,322 unmonitored patients, 370 achieved ROSC at the scene. Only six (0.6%) of the 952 who did not achieve ROSC at the scene survived, and all six were readily identifiable as having persistent ventricular fibrillation. Excluding those patients with persistent ventricular fibrillation, all survivors achieved ROSC within 25 minutes after paramedic arrival. CONCLUSIONS: Excluding patients with persistent ventricular fibrillation, resuscitative efforts can be terminated at the scene when normothermic adults with unmonitored, out-of-hospital, primary cardiac arrest do not regain spontaneous circulation within 25 minutes following standard advanced cardiac life support. These criteria should now be validated in several large centers with high survival rates.

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Predicting the Outcome of Unsuccessful Prehospital Advanced Cardiac Life Support—AL Kellermann, BB Hackman, G Somes. JAMA 1993;270(12):1433.

OBJECTIVE: To determine if failure to achieve return of spontaneous circulation following prehospital advanced cardiac life support (ACLS) warrants termination of efforts at the scene. DESIGN: Retrospective case series. SETTING: Memphis, Tennessee, a city of 610,337 people that is served by a fire department-based emergency medical service system. All city ambulances provide ACLS. PATIENTS: Adult victims of out-of-hospital cardiac arrest due to heart disease. INTERVENTION: All patients received prehospital ACLS according to the 1986 American Heart Association guidelines. Following prehospital ACLS, all patients were transported to the nearest hospital emergency department whether or not a pulse was restored in the field. MAIN OUTCOME MEASURES: Survival to hospital admission, survival to hospital discharge, and neurological status at discharge. RESULTS: Over the 39-month study interval, the Memphis Fire Department treated 1,068 victims of out-of-hospital cardiac arrest. Three hundred ten of these (29%) had return of spontaneous circulation prior to transport for some period. The remaining 758 patients (71%) never regained a pulse and were transported with ongoing cardiopulmonary resuscitation. Patients who had return of spontaneous circulation prior to transport were more likely to be admitted (69% vs 7.0%) and far more likely to be discharged alive (26.5% vs 0.4%) than patients who failed to respond to prehospital ACLS. Three patients who survived to hospital discharge despite failure to achieve return of spontaneous circulation prior to emergency medical service transport sustained their cardiac arrest after paramedic arrival. All three were discharged with moderate to severe cerebral disability. CONCLUSIONS: Rapid transport of adults who fail to respond to an adequate trial of prehospital ACLS does not result in meaningful rates of survival. In such cases, on-line emergency medical service physicians should authorize paramedics to cease efforts in the field.

Modes of Death in the Pediatric Intensive Care Unit: Withdrawal and Limitation of Supportive Care—DD Vernon, JM Dean, OD Timmons, W Banner Jr, EM Allen-Webb. Crit Care Med 1993;21(11): 1798.

OBJECTIVE: To determine the frequency of withdrawal or limitation of supportive care for children dying in a pediatric intensive care unit (ICU). DESIGN: Retrospective review of medical records. SETTING: Pediatric ICU in a tertiary care children's hospital. PATIENTS: All children dying in the pediatric ICU over a 54-month period (n = 300). INTERVENTIONS: Medical record review. MEASUREMENTS & MAIN RESULTS: Data recorded for each patient included diagnosis, mode of death, and whether the child was brain dead. Each patient was assigned to one of the following mode of death categories: brain dead; active withdrawal of supportive care (meaning removal of the endotracheal tube); failed cardiopulmonary resuscitation; allowed to die without cardiopulmonary resuscitation (do-not-resuscitate status). A total of 300 patients were identified. Diagnoses included postoperative congenital heart disease (n = 56), head trauma (n = 38), near-miss sudden infant death syndrome (n = 28), pneumonia (n = 22),

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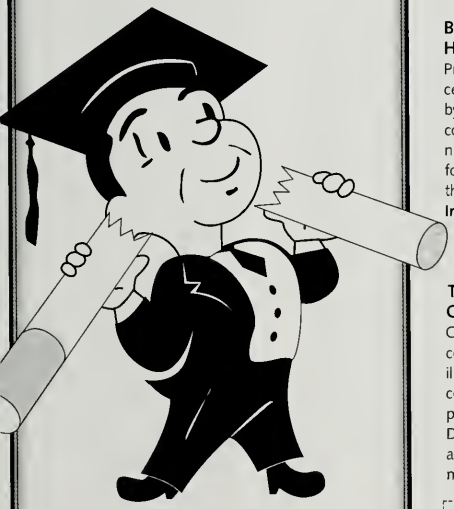

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sepsis (n = 21), near-drowning (n = 21), various anoxic insults (n = 20), multiple trauma (n = 17), and patients with other diagnoses (n = 77). Mode of death was active discontinuation of support in 95 (32%) patients, do-not-resuscitate status in 78 (26%), brain death in 70 (23%), and failed cardiopulmonary resuscitation in 57 (19%). CONCLUSIONS: In a large, multidisciplinary pediatric ICU, the most common mode of death was active withdrawal of support. In addition, more than half (173/300, 58%) of children dying in the pediatric ICU underwent either active withdrawal or limitation (do-not-resuscitate status) of supportive care.

Free Radicals and Antioxidants in Sepsis—HF Goode, NR Webster. Crit Care Med 1993;21(11):1770.

OBJECTIVES: The clinical condition of sepsis is caused by the release of numerous mediators from many cells. The purpose of this review is to describe the results of studies in which the role of free radicals and/or the potential therapeutic value of antioxidants are assessed. DATA SOURCES: The studies described in this review come from a variety of sources, including Med-Line CD-ROM computerized database, Index Medicus, and references identified from the bibliographies of pertinent articles and books. Reports were confined to English language articles from 1966 to 1992. STUDY SELECTION: All retrieved references in which free-radical activity was assessed or antioxidants were measured or administered in sepsis or endotoxemia were included. This selection process encompassed clinical, animal, and in vitro cell culture work. DATA EXTRACTION: Cited literature was found in reputable peer-reviewed clinical or basic science journals. DATA SYNTHESIS: Any contradictions in the results of studies are discussed. CONCLUSIONS: There is evidence that free radicals play an important role in the pathogenesis of sepsis. Antioxidant therapy has the potential to protect against such injury. It is suggested that combination therapy, which augments the endogenous antioxidant defenses, is likely to be the best approach.

The Anencephalic Organ Donor: A Challenge to Existing Moral and Statutory Laws—JH Diaz. Crit Care Med 1993;21(11):1781.

OBJECTIVES: To inform physicians of the critical shortage of neonatal heart donors and to provide arguments for and against selecting brain-absent anencephalics as heart donors for brain-normal infants who are dying of hypoplastic left heart syndrome. DATA SOURCES: International scientific journals of medicine, genetics, epidemiology, bioethics, and public health; adjudicated U.S. civil court cases; and state regulations and statutes. STUDY SELECTION: Worldwide experience with anencephalics as homologous organ donors. DATA EXTRACTION: Demographic and epidemiologic data on anencephalic births and natural histories; U.S. civil, district, and appellate court case decisions directing or prohibiting organ donation; state determination-of-death acts; state uniform-anatomical-gift acts. DATA SYNTHESIS: Organization of all data into either moral challenges or legal challenges to anencephalic organ donation. CONCLUSIONS: Statutory laws pose a greater challenge to anencephalic organ donation than moral laws. Case law reviews eliminate substituted judgment rulings in directing anencephalic organ donation. A redefinition of brain death applying only to human beings born without a brain would make more donor hearts available to brain-normal infants dying of hypoplastic left heart syndrome.

A Preliminary Study of Cardiopulmonary Resuscitation by Circumferential Compression of the Chest with Use of a Pneumatic Vest—HR Halperin, JE Tsitlik, M Gelfand, ML Weisfeldt, KG Gruben, HR Levin, et al. N Engl J Med 1993; 329:762.

BACKGROUND: More than 300,000 people die each year of cardiac arrest. Studies have shown that raising vascular pressures during cardiopulmonary resuscitation (CPR) can improve survival and that vascular pressures can be raised by increasing intrathoracic pressure. METHODS: To produce periodic increases in intrathoracic pressure, we developed a pneumatically cycled circumferential thoracic vest system and compared the results of the use of this system in CPR (vest CPR) with those of manual CPR. In Phase 1 of the study, aortic and right-atrial pressures were measured during both vest CPR (60 inflations per minute) and manual CPR in 15 patients in whom a mean (± SD) of 42 ± 16 minutes of initial manual CPR had been unsuccessful. Vest CPR was also carried out on 14 other patients in whom pressure measurements were not made. In Phase 2 of the study, short-term survival was assessed in 34 additional patients randomly assigned to undergo vest CPR (17 patients) or continued manual CPR (17 patients) after initial manual CPR (duration, 11 ± 4 minutes) had been unsuccessful. RESULTS: In Phase 1 of the study, vest CPR increased the peak aortic pressure from 78 ± 26 mm Hg to 138 ± 28 mm Hg ($p < 0.001$) and the coronary perfusion pressure from 15 ± 8 mm Hg to 23 ± 11 mm Hg ($p < 0.003$). Despite prolonged unsuccessful manual CPR, spontaneous circulation returned with vest CPR in 4 of the 29 patients. In Phase 2 of the study, spontaneous circulation returned in 8 of the 17 patients who underwent vest CPR as compared with only 3 of the 17 pa-

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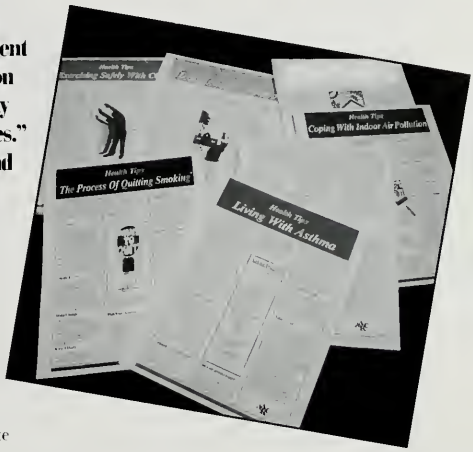

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tients who received continued manual CPR ($p = 0.14$). More patients in the vest-CPR group than in the manual-CPR group were alive 6 hours after attempted resuscitation (6 of 17 vs 1 of 17) and 24 hours after attempted resuscitation (3 of 17 vs 1 of 17), but none survived to leave the hospital. CONCLUSIONS: In this preliminary study, vest CPR, despite its late application, successfully increased aortic pressure and coronary perfusion pressure, and there was an insignificant trend toward a greater likelihood of the return of spontaneous circulation with vest CPR than with continued manual CPR. The effect of vest CPR on survival, however, is currently unknown and will require further study.

Assessment of Splanchnic Oxygenation by Gastric Tonometry in Patients with Acute Circulatory Failure—N Maynard, D Bihari, R Beale, M Smithies, G Baldock, R Mason, I McColl. JAMA 1993;270 (10):1203.

OBJECTIVE: To investigate the importance of splanchnic ischemia in patients with acute circulatory failure by comparing gastric intramucosal pH as measured by tonometry with conventional methods of assessing adequacy of tissue oxygenation. DESIGN: Prospective cohort of patients with acute circulatory failure in first 24 hours after admission to the intensive care unit. SETTING: Two general intensive care units in London, England. PATIENTS: Consecutive sample of 83 patients of varying diagnostic categories that required pulmonary artery catheterization. MAIN OUTCOME MEASURES: Gastric intramucosal pH and hemodynamic, oxygen transport, and metabolic variables were measured on admission and at 12 hours and 24 hours after admission. Prediction of outcome (death or survival) by each measurement was assessed by sensitivity, specificity, and logistic regression analysis. RESULTS: Mean 24-hour Acute Physiology and Chronic Health Evaluation (APACHE II) score was 20.3. There were significant differences in mean gastric intramucosal pH between survivors and nonsurvivors on admission and at 24 hours (7.40 vs 7.28, 7.40 vs 7.24, respectively; $p < 0.001$). Admission heart rate was higher (116 vs 101 beats per minute; $p < 0.003$) and mean arterial pressure lower (82 vs 97 mm Hg; $p < 0.01$) in nonsurvivors. There were no consistent differences in cardiac index, oxygen delivery, and oxygen uptake between survivors and nonsurvivors. Admis-sion arterial pH was significantly lower (7.3 vs 7.36; $p < 0.003$), base excess more negative (–5.3 vs –1.9; $p < 0.001$), and lactate concentration higher (3.14 vs 1.91 mmol/L; $p < 0.03$) in nonsurvivors. Gastric intramucosal pH had a sensitivity of 88% for predicting death and a likelihood ratio of 2.32, higher than for any other variable. Only gastric intramucosal pH at 24 hours independently predicted outcome. CONCLUSIONS: Gastric intramucosal pH was the most reliable indicator of adequacy of tissue oxygenation in this group of patients. Inadequate regional blood flow as detected by a reduction in gastric intramucosal pH, but not by systemic measures, is an important contributor to morbidity and mortality in intensive care units.

Endotracheal Intubation and Mechanical Ventilation in Severe Asthma—JL Zimmerman, RP Dellinger, AN Shah, RW Taylor. Crit Care Med 1993;21(11):1727.

OBJECTIVE: To determine the occurrence rate of complications and mortality in patients with severe asthma requiring endotracheal intubation and mechanical ventilation. DESIGN: Retrospective review of medical records from September 1982 to July 1988. SETTING: Urban, teaching hospital serving primarily indigent patients. PATIENTS: Fifty-seven adult patients with asthma requiring tracheal intubation and mechanical ventilation. INTERVENTIONS: None. MEASUREMENTS & MAIN RESULTS: Fifty-seven patients requiring tracheal intubation and mechanical ventilation during 69 hospital admissions were identified. Medication noncompliance and upper respiratory tract infections were recorded as the most frequent precipitating events for exacerbation of asthma. Forty-nine intubations were initiated because of a clinical diagnosis of respiratory distress, but multiple indications were present in 42 admissions. One or more complications occurred in 31 episodes of endotracheal intubation and mechanical ventilation (45%). Death occurred in four (6%) of 69 admissions. Three of the four deaths occurred in patients who had a cardiorespiratory arrest before hospital admission. CONCLUSIONS: While complications occurred in 45% of patients with severe asthma requiring intubation and mechanical ventilation, the mortality rate was low. We conclude that intubation and mechanical ventilation in patients with life-threatening asthma are safe and beneficial interventions.

Disposition and Respiratory Effects of Intrathecal Morphine in Children—DG Nichols, M Yaster, AM Lynn, MA Helfaer, JK Deshpande, PN Manson, et al. Anesthesiology 1993;79(4):733.

BACKGROUND: The extent and duration of respiratory depression after opioid administration are poorly defined in infants and children. METHODS: The disposition and respiratory effects of intrathecal morphine were studied in 10 patients (ages 4 mo to 15 yr) after repair of craniofacial defects. Morphine, 0.02 mg/kg, was administered intrathecally before the

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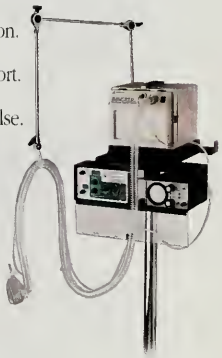

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end of surgery. Postoperatively, we determined the minute ventilation (V_E) in response to increasing partial pressure of end-tidal carbon dioxide (P_{ETCO_2}) during carbon dioxide rebreathing. The slope (V_E/P_{etCO_2}) and intercept (V_E at P_{etCO_2} 60 mm Hg, V_E 60) of the carbon dioxide response curve were calculated at 6, 12, and 18 h after morphine administration. Cerebrospinal fluid (CSF) and blood were analyzed for morphine concentration by radioimmunoas-say. RESULTS: Mean V_E/P_{etCO_2} decreased from a preoperative value of 35.1 ± 3.7 to 16.3 ± 2.8 $mL \cdot kg^{-1} \cdot min^{-1} \cdot mm\ Hg^{-1}$ at 6 h after morphine, and remained depressed to 23.4 ± 2.9 and 23.5 ± 3.3 $mL \cdot kg^{-1} \cdot min^{-1} \cdot mm\ Hg^{-1}$ at 12 h and 18 h, respectively, compared to preoperatively). The infants' (n = 3) V_E/P_{etCO_2} at 6 h were 21, 4, and 27 $mL \cdot kg^{-1} \cdot min^{-1} \cdot mm\ Hg^{-1}$. Mean V_E 60 decreased from 874 ± 125 to 276 ± 32 $mL \cdot kg^{-1} \cdot min^{-1}$ at 6 h, but then recovered at 12 and 18 h to 491 ± 68 and 567 ± 82 $mL \cdot kg^{-1} \cdot min^{-1}$, respectively. The infants' V_E 60 at 6 h were 350, 142, and 245 $mL \cdot kg^{-1} \cdot min^{-1}$. Mean CSF morphine concentration was 2,860 ± 540 ng/mL at 6 h, and decreased to 640 ± 220 and 220 ± 150 ng/mL at 12 and 18 h, respectively. CONCLUSIONS: Intrathecal morphine, 0.02 mg/kg, depressed the ventilatory response to carbon dioxide for up to 18 h concomitant with increased CSF morphine concentrations. Infants (4-12 months of age) did not exhibit greater ventilatory depression than did children (2-15 yr of age).

Ventilatory Drive and Carbon Dioxide Response in Ventilatory Failure Due to Myasthenia Gravis and Guillain-Barré Syndrome—CO Borel, JS Teitelbaum, DF Hanley. Crit Care Med 1993;21(11): 1717.

OBJECTIVE: To test the hypothesis that either decreased ventilatory drive or decreased CO_2 responsiveness accounts for the hypoventilation observed in patients during acute ventilatory failure from myasthenia gravis or Guillain-Barré syndrome. DESIGN: Prospective, consecutive case series evaluating trials of ventilatory muscle performance, ventilatory drive, and CO_2 response in patients during recovery from ventilatory failure until they were weaned from mechanical ventilation. SETTING: Neurosciences critical care unit in a university hospital. PATIENTS: Seven intubated, mechanically ventilated patients with myasthenia gravis or Guillain-Barré syndrome. INTERVENTIONS: Patients repeatedly performed mechanically unsupported, spontaneous breathing trials to the limits of endurance. After spontaneous breathing trials, patients underwent CO_2 rebreathing studies. MEASUREMENTS & MAIN RESULTS: Seventy-three breathing trials were performed in three patients with Guillain-Barré syndrome and four patients with myasthenia gravis. Patients were unable to sustain spontaneous ventilation in 55 trials averaging 27 ± 5 mins. In these trials, significant increases occurred in mean end-tidal CO_2 (41 ± 1 to 44 ± 1 torr [5.6 ± 0.1 to 6.0 ± 0.1 kPa]) and respiratory rate (31 ± 1 to 35 ± 1 breaths/min, $p < 0.01$). Ventilatory drive (as measured by airway occlusion pressure for 100 msecs) increased significantly $p < 0.01$ from 3.7 ± 0.3 to 4.9 ± 0.3 cm H_2O. The response of airway occlusion pressure to CO_2 rebreathing after these trials was 0.33 ± 0.07 cm H_2O/sec/mm Hg, while the minute ventilation response to CO_2 rebreathing was only 0.30 ± 0.06 L/min/mm Hg. CONCLUSIONS: These results suggest that ventilatory drive increases during acute hypoventilation, and the ventilatory drive response to CO_2 remains intact, even when the minute ventilation response to CO_2 is poor. Therefore, a decrease in ventilatory drive or CO_2 response is unlikely to account for hypoventilation during ventilatory failure in patients with myasthenia gravis or Guillain-Barré syndrome.

Absence of Nonshivering Thermogenesis in Anesthetized Adult Humans—JM Hynson, DI Sessler, A Moayeri, J McGuire. Anesthesiology 1993;79(4):695.

BACKGROUND: Typically, core temperature rapidly decreases after induction of anesthesia, but reaches a stable plateau after several hours. This plateau typically occurs in conjunction with the onset of thermoregulatory vasoconstriction. Decreased heat loss, caused by vasoconstriction, may not be sufficient to establish thermal steady state without a concomitant increase in heat production. Accordingly, the authors tested the hypothesis that nonshivering thermogenesis contributes to thermal steady state during anesthesia. Rewarming from hypothermia is often associated with an afterdrop (a further reduction in core temperature, despite cutaneous warming). Because total body heat content increases during cutaneous warming, heat storage during afterdrop must reflect increased temperature and heat content of the peripheral tissue mass. Thermal balance was measured during rewarming to estimate the thermal capacity of the peripheral tissues. METHODS: Five volunteers were anesthetized with isoflurane and paralyzed with vecuronium. Oxygen consumption was measured during cooling to a core temperature at least 1° C less than that which triggered vasoconstriction. Volunteers were subsequently rewarmed using a circulating-water blanket and forced-air warmer. Oxygen consumption and cutaneous heat flux were measured to assess thermal balance and peripheral tissue heat storage during rewarming. RESULTS: The core temperature threshold for vasoconstriction was

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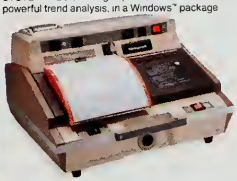

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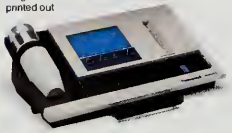

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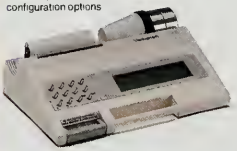

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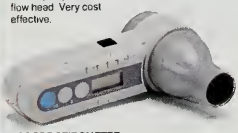

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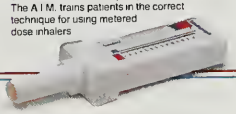

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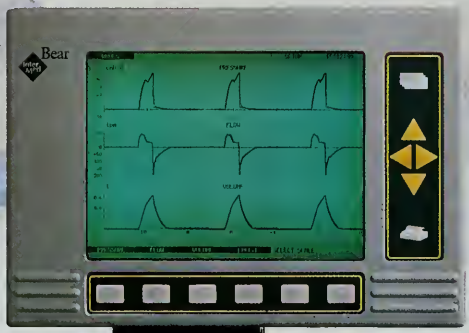

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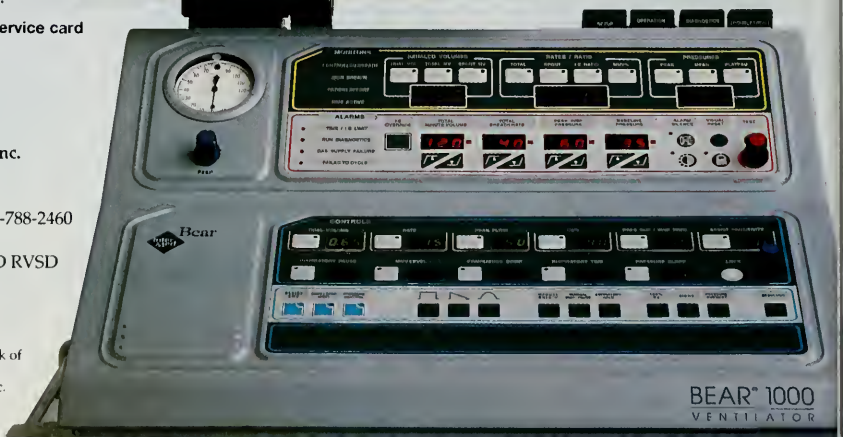

35.2 ± 0.8° C. Oxygen consumption decreased 9 ± 5%/° C during active cooling before vasoconstriction and 9 ± 3%/° C after vasoconstriction. After the start of rewarming, core temperature continued to decrease for an additional 32 ± 8 min. The magnitude of this afterdrop was 0.6 ± 0.1° C. Peripheral tissue heat storage measured from the start of rewarming until the first net increase in core temperature was 144 ± 60 kcal, which approximately equals 2 h of resting metabolic heat production. CONCLUSIONS: The authors concluded that nonshivering thermogenesis is not an important thermoregulatory response in adults anesthetized with isoflurane. Afterdrop and delayed core temperature recovery during rewarming reflect the large heat storage capacity of peripheral tissues.

Relationship between Oxygen Uptake and Oxygen Delivery in Septic Patients: Effects of Prostacyclin versus Dobutamine—D De Backer, J Berré, H Zhang, RJ Kahn, J-L Vincent. Crit Care Med 1993;21(11):1658.

OBJECTIVE: To compare the effects of prostacyclin (PGI_2) and dobutamine on the relationship between oxygen delivery ($\dot{D}_{O_2}$) and oxygen uptake ($\dot{V}_{O_2}$) in stable septic patients. DESIGN: Prospective study using a crossover design with alternate order of medications. PATIENTS: Seventeen patients with documented sepsis and a stable hemodynamic status with normal blood lactate concentrations. Eleven patients were mechanically ventilated. Eight patients eventually died. INTERVENTIONS: $\dot{D}_{O_2}$ and $\dot{V}_{O_2}$ were calculated before and after a 20-min infusion of 5 ng/kg/min of PGI_2 followed or preceded by 5 μg/kg/min of dobutamine. MEASUREMENTS & MAIN RESULTS: Both medications increased cardiac output significantly. At the dose used, PGI_2 infusion reduced mean arterial pressure from 90.8 ± 16.8 to 81.5 ± 17.3 mm Hg ($p < 0.01$) and P_{aO_2} from 97 ± 25 torr to 82 ± 22 torr (from 12.9 ± 3.3 to 10.9 ± 2.9 kPa) ($p < 0.01$) and increased venous admixture from 17.5 ± 0.6% to 23.8 ± 8.2% ($p < 0.01$). Dobutamine had no significant influence on these variables. PGI_2 increased $\dot{D}_{O_2}$ by 19% (from 470 ± 105 to 557 ± 117 mL/min/m^2, $p < 0.01$) while dobutamine increased $\dot{D}_{O_2}$ by 27% (from 463 ± 103 to 589 ± 156 mL/min/m^2, $p < 0.01$). PGI_2 increased $\dot{V}_{O_2}$ by 5% (from 148 ± 38 to 155 ± 36 mL/min/m^2) while dobutamine increased $\dot{V}_{O_2}$ by 10% (from 146 ± 36 to 161 ± 41 mL/min/m^2, $p < 0.01$). Accordingly, there was an identical decrease in oxygen extraction with PGI_2 (from 32.4 ± 8.2% to 28.6 ± 7.1%, $p < 0.01$) and dobutamine (from 32.4 ± 8.3% to 28.5 ± 7.8%, $p < 0.01$). The responses to these medications were similar in survivors and nonsurvivors. CONCLUSIONS: PGI_2 and dobutamine at the doses used have similar effects on oxygen extraction in critically ill, septic patients, but dobutamine increases $\dot{D}_{O_2}$ more consistently and is better tolerated than prostacyclin.

Effects of Prophylactic Antiarrhythmic Drug Therapy in Acute Myocardial Infarction: An Overview of Results from Randomized Controlled Trials—KK Teo, S Yusuf, CD Furberg. JAMA 1993;270 (13):1589.

OBJECTIVE: To investigate the effects of prophylactic therapy with antiarrhythmic agents on mortality in patients with myocardial infarction. DATA SOURCES & STUDY SELECTION: Data were obtained from all completed, published or unpublished, randomized, parallel controlled trials of antiarrhythmic agents, regardless of sample size. Investigators were contacted for data on patients excluded after randomization. DATA EXTRACTION: Data on mortality were extracted by one author and confirmed where necessary by the others. DATA SYNTHESIS: Mortality data from 138 trials on 98,000 patients were combined by the Yusuf-Peto adaptation of the Mantel-Haenszel method. There were 660 deaths among 11,712 patients allocated to receive Class I agents and 571 deaths among 11,517 corresponding control patients (51 trials: odds ratio [OR], 1.14; 95% confidence interval [CI], 1.01 to 1.28; p = 0.03). Of 26,973 patients allocated to receive ß-blockers (Class II agents), 1,464 died compared with 1,727 deaths among 26,295 control patients (55 trials: OR, 0.81; 95% CI, 0.75 to 0.87; p = 0.00001). Of 778 patients allocated to receive amiodarone (a Class III agent), 77 died compared with 101 deaths in 779 control patients (eight trials: OR, 0.71; 95% CI, 0.51 to 0.97; p = 0.03). There were 982 deaths in 10,154 patients allocated to receive a Class IV agent (calcium channel blockers) and 949 deaths in 10,188 control patients (24 trials: OR, 1.04; 95% CI, 0.95 to 1.14; p = 0.41). CONCLUSIONS: The routine use of Class I antiarrhythmic agents after myocardial infarction is associated with increased mortality. ß-blockers have been conclusively demonstrated to reduce mortality. The limited data on amiodarone appear promising. Data on calcium channel blockers remain unpromising.

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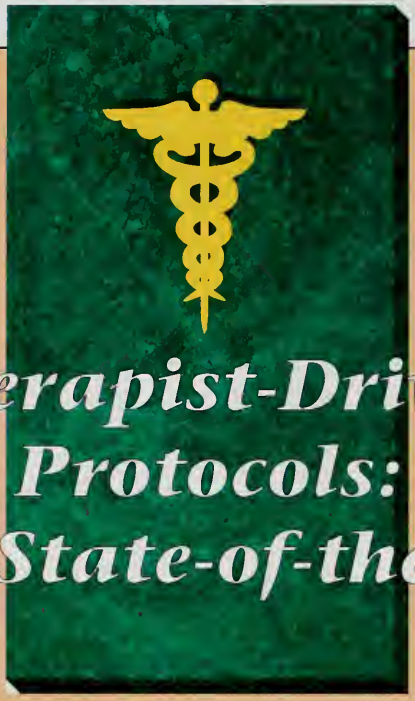

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CONTROVERSIES IN HOME RESPIRATORY CARE

Part I

A Special Issue

based on a conference
held October 29-31, 1993
in St Petersburg Beach, Florida

sponsored by
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Sam P Giordano MBA RRT
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Nicholas S Hill MD
Leslie Hoffman RN PhD
Robert M Kacmarek PhD RRT
Patrick Leger MD
Barry Make MD
David J Pierson MD
James K Stoller MD

Discussants: The chairmen, the speakers, Robert Campbell RRT, Doug McIntyre RRT, Paul J Mathews MPA RRT, Ray Masferrer RRT, Homer Rodriguez RRT, Susan Sortor-Leger RRT, and Kay Donna Stelmach RN RRT

1 Karen A Hardy
Patrick J Dunne
2. Michael J Decker
3. James K Stoller
David J Pierson
4. Robert S Campbell
5. Paul J Mathews
6. Bartolome R Celli
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11. John R Bach
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16 Homer Rodriguez
Kaye Weber
17 Sam P Giordano

Group Photo (left to right)

Back Row: Ray Masferrer, Eugene G Fletcher, Elizabeth S Marson, Leslie A Hoffman, Robert M Kacmarek, Bartolome Celli, Patrick Leger, Homer Rodriquez, Doug McIntyre, Paul J Mathews.

Middle Row: John R Bach, Nicholas S Hill, Mary E Gilmartin, James K Stoller, Karen A Hardy, Pat Brougher, Barry Make, Patrick J Dunne

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Dear Reader:

The American Association for Respiratory Care and its science journal RESPIRATORY CARE are proud to present the April and May 1994 issues containing the proceedings from the 1993 Journal Conference "Controversies in Home Respiratory Care." The papers and discussions presented here are well-organized state-of-the-art materials designed for you to use in your daily practice.

Considerable planning and work are required to present and publish the proceedings of a conference like this. We are grateful to the speakers, the Editorial Board, Conference Co-Chairmen Robert Kacmarek and James K Stoller, Editor Pat Brougher, Associate Editor Kaye Weber, and Assistant Editor Donna Stephens for planning, writing, and editing these issues and for participating in the Conference and sharing this important information with us.

We are indebted to Allen & Hanburys, Division of Glaxo Inc, for their generous grant to the Association, which made the presentation and publication of the Conference proceedings possible. Allen & Hanburys has supported the last six Journal Conferences—for this we are enormously grateful.

Sincerely,

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Managing Editor
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Controversies in Home Respiratory Care: Conference Summary

David J Pierson MD

Introduction

This summary introduces 15 articles on home respiratory care, each written by a recognized authority on the topic discussed.[1-15] The conference at which these papers were presented was not intended to be a consensus conference, but was instead a state-of-the-art exercise to identify and review the most important areas within this field. However, in the course of two and one-half days of presentations and discussions, a remarkable amount of consensus became evident. In this article, I attempt to summarize that consensus for each of the main topic areas addressed during the conference, and also to identify areas in which additional study could be most helpful.

Main Messages of the Conference

Table 1 lists what I consider to be the most important take-home messages of this conference—the key issues in home respiratory care today—although I have not listed them in any particular order of priority. First is the importance of shifting from an *equipment focus* to an *education- and care-coordinating focus* in home respiratory care. This applies not only to the in-home activities themselves, but also (and especially) to the respiratory care practitioner. The field of respiratory care still suffers from the legacy of the 1960s and early 70s, and its associations with intermittent positive-pressure breathing (IPPB) and the other poorly documented techniques to which we hitched our fortunes, and as long as we are tied primarily to the *equipment* in the home (as well as that in the intensive care unit), I think everyone involved in respiratory care suffers. The respiratory care practitioner needs to become an educator, a consultant, and a coordinator of care, and less a person installing or operating a medical device. I recognize the difficulties posed by current reimbursement mechanisms, tied as they are to equipment rather than to patient care and other services by respiratory care practitioners, as we work to act on this first message. Clearly, bringing these funding mechanisms more into line with the realities of home care continues to be an extremely important goal.

Table 1. Key Issues in Home Respiratory Care Emerging from 1993 Journal Conference

1. Importance of shifting from equipment focus to education- and care-coordinating focus
2. Central importance of the caregiver in the home
3. Collaborative self-management as the preferred approach
4. Importance of the cultural context in home care
5. Quality of life as the most important goal and/or outcome in home care
6. Importance of cost-benefit in all aspects of home care

Second is the importance of *the caregiver in the home*, by whom I mean the person who is there most of the time actually assisting the patient. That

Dr Pierson is Medical Director, Respiratory Care, Harborview Medical Center, and Professor of Medicine, University of Washington, Seattle, Washington.

A version of this summary was presented following the RESPIRATORY CARE Journal Conference "Controversies in Home Respiratory Care," held in St Petersburg Beach, Florida, October 29-31, 1993.

Reprints: David J Pierson MD, Harborview Medical Center, Respiratory Care ZA-62, 325 Ninth Ave, Seattle WA 98104.

is usually someone other than the patient, particularly in long-term mechanical ventilation, but it might be the patient him- or herself, for example, with long-term oxygen therapy (LTOT) or continuous positive airway pressure (CPAP). More and more this caregiver is a lay person such as a family member or other individual trained to care for that particular patient in the home, rather than a high-priced, high-expertise healthcare professional such as a home-care nurse. Obviously, this issue is linked to Item Number 1 because a key aspect of the role of supervisor, coordinator, and educator is working with people with less formal training, whose services produce less of an economic drain on the healthcare system.

The third main message of this conference is that *collaborative self-management* should be a primary goal for patients with respiratory disease who are cared for at home. I was unfamiliar with that terminology prior to Barry Make's presentation,[9] but it is obviously integral to all the other issues in Table 1 and to any lasting significance that may be derived from this conference. I think all the participants would readily agree that wherever it is possible and to the degree to which it is possible, enlightened self-management in collaboration with the providers is the preferred approach in every instance—not always achievable, but the preferred approach.

Fourth is the importance of the *cultural context* in home care. The approaches and therapeutic modalities discussed at the conference and in the papers comprising these two special issues of RESPIRATORY CARE essentially describe home care that has been set up for, applied to, and studied in middle-class Americans of northern European heritage. That is a fact of American healthcare, and, of course it exists throughout the system and not just in home respiratory care.

But, it is also a fact of American healthcare that our cultural diversity is increasing rapidly and that with many of our citizens today, the models that we have used are simply not going to work. In my hospital, we have on-call interpreter services for more than 70 languages, and these services are used every day for at least a dozen of them. Many of the patients who speak those languages come from backgrounds so different from those of the people who are in control of healthcare in this country (such as the participants in this conference) that the care offered to those people is unlikely to be very beneficial unless the relevant cultural context is taken into account. That means that collaborative self-management may not always be the right model for a given patient.

The cultural diversity that I have described exists just within the United States, and as respiratory care becomes international and worldwide, as is happening under the leadership of the American Association for Respiratory Care (AARC) and a number of the participants in this conference, the relationship between Issues 3 and 4 in Table 1 becomes increasingly important.

Fifth on the list is the concept of *quality of life as the most important goal* or outcome in home care. Several of the topics discussed at the conference—such as home oxygen therapy and long-term mechanical ventilation—have been assessed in terms of their impact on survival. We have talked about survival as the gold standard, as something that is unequivocally measurable. Yet I think everyone at the conference would agree that an increase in survival per se should not be the primary goal of home care. Instead, even if the patient's life is not extended by 1 hour, if they are able to live a better life—that is, if quality of life is improved—the most important goal of care has been achieved.

However, even quality of life cannot be considered apart from costs (Item 6 in Table 1), and thus home care must deal increasingly with *cost-benefit* considerations. I'm afraid we can't escape this increasing focus on cost, something that will surely be a main determinant of the future of respiratory home care.

Home Care in the Contexts of Quality of Life and Cost Benefit

Quality of Life and the Healthcare Continuum

One of the most important things to be presented at this conference is the concept of the healthcare continuum, which depicts the different sites and intensities of care along a continuum from the most intensive (the acute-care hospital), through the available intermediate options, to the least intensive (home care). This concept is discussed in more detail by both Patrick Dunne[1] and Sam Giordano[2] in

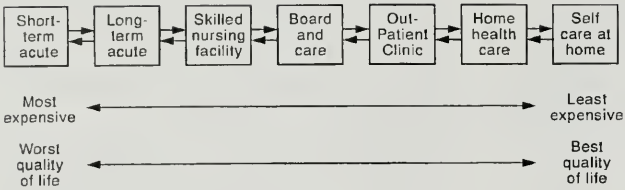

Fig. 1. The healthcare continuum as discussed at this conference, considered in the contexts of cost and quality of life for the patient.

their articles elsewhere in this issue. My conception of the major thrust of this conference can be illustrated using the variations on this continuum concept that are shown in Figures 1 and 2.

Figure 1 shows the array of care sites and/or approaches currently used in the United States. If we imagine a patient requiring technology-intensive respiratory care (for example, ventilatory assistance), the 7 boxes depict short-term acute care on the far left and self-care on the far right, representing progressive stages in the degree of regulation, the overall expense, the intensity of effort, and also the patient's quality of life (although this was not originally included). Intensity (and cost) of care and quality of life have a generally inverse relationship, as suggested by the arrows below the boxes. These arrows go from the most expensive all the way to the least expensive and from the worst quality of life (thinking here, for example, of the ventilator-dependent patient who is no longer acutely ill but remains in an intensive care unit) to the best quality of life (self-care at home).

I added Figure 2 because the more I looked at Figure 1 the more I felt it was incomplete from the quality-of-life point of view. In informal discussion at one of the breaks during the conference, someone remarked on how different rehabilitation centers tend to be from long-term ventilator units, in terms of what the patients actually *do* most of the time. Walking through many long-term acute-care facilities one sees patients lying in beds, just like in an intensive care unit. But walking through the typical rehabilitation center, one finds the patients up, out of bed, doing things. I cannot help concluding that there is a difference in what the patients are experiencing—a difference in quality of life.

This difference is shown in Figure 2 by means of the two axes—cost on the vertical and quality of life on the horizontal. Patients get a lot farther out on the quality of life scale with the track that takes them toward restoration of independent living and increased function. I don't see increased function and the restoration of independent living as primary goals of the steeper, long-term acute-care track that leads into a skilled nursing home or, if the patient is lucky, into a less medically intensive nursing home or some other custodial arrangement, which may cost society less but can hardly be expected to improve quality of life very much. Clearly, the separation between the long-term acute-care track and the rehabilitative track is less obvious in many instances than I have shown it in the figure, but the concept is important. In all the subject areas discussed in the papers and discussions comprising these two special issues of the Journal, quality of life should be kept in the forefront, more so than it has tended to be in the past.

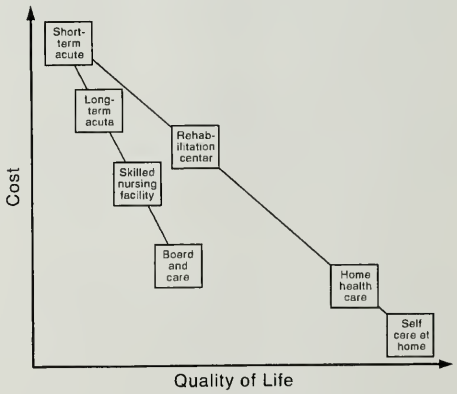

Fig. 2. Another view of the healthcare continuum, its components slightly modified, shown in relation to cost and quality of life. The axes are qualititative rather than quanititative, but the relative positions of the different sites and care schemes are consistent with the consensus reached at the conference.

Assessing Cost-Benefit in the Context of Quality of Life

In an attempt to be more precise in evaluating the treatment modalities covered in this conference, I have devised an arbitrary but quasi-objective scheme for focusing our attention on cost-benefit and quality of life (Table 2). This scheme derives a sort of cost-benefit ratio to apply to each of the therapies and techniques discussed, and later I rate them all in order to emphasize the importance of cost-benefit and quality of life in all the currently important areas of home respiratory care.

Table 2. An Arbitrary Rating Scheme for Benefits and Costs associated with Home Respiratory Care*

Category	Score	Description
Benefit	5	Increases quality of life, increases survival
	4	Increases quality of life, increased survival not demonstrated
	3	Reduces need for hospitalization, reduces other costs
	2	Objective benefit (by any measure)
	1	Subjective benefit, use supported by rationale or inference from other settings
Cost†	5	> $10,000/month
	4	$1,000 - $10,000/month
	3	$100 - $1,000/month
	2	$10 - $100/month
	1	< $10/month

*If the benefit-cost ratio exceeds 1, the technique or modality is cost-effective; if the ratio is less than 1, it is not cost-effective.

†This cost score increases by 1 point if the technique or modality involves substantial discomfort, risk, or 'hassle' for the patient or caregiver.

In keeping with the goal of improved quality of life as the most important objective, the scheme assigns 5 points to something that increases survival and also improves quality of life. A good example would be long-term oxygen therapy for patients with chronic obstructive pulmonary disease (COPD) and chronic hypoxemia, begun at the right time and in the appropriate clinical setting. Something that increases the quality of a patient's life, even if it does not prolong survival, gets 4 points. In this era of healthcare reform based in large part on cost, a modality that reduces the need for hospitalization and other expensive forms of care is obviously worthwhile and would be assigned 3 points on the 'benefit' scale.

A large proportion of the medical literature focuses on acute, short-term changes, often in some measured or derived physiologic parameter, in response to a given therapy or procedure. In such cases there is objective benefit, although any relationship with longer-term outcomes or quality of life tends to be inferred if it is considered at all. For such interventions, the scheme awards 2 points. Finally, if all we have is the impression that a treatment helps, even in the face of a strong physiologic basis or other compelling rationale, it is awarded only 1 point on the benefit scale.

For the cost axis, I arbitrarily used a logarithmic progression through the range into which the things discussed at this conference fall in terms of dollars/month. An actual cost exceeding $10,000/month is awarded 5 points; between $1,000 and $10,000, 4 points; $100 to $1,000, 3 points; and $10 to $100/month, 2 points. Examples in the first three categories would be home mechanical ventilation for an infant who requires continuous nursing care, part-time ventilatory support for an adult receiving frequent in-home visits who is assisted mainly by a family member, and continuous oxygen therapy for an ambulatory patient, respectively. Therapies or techniques that cost less than $10/month, such as the use of modified breathing patterns or regular exercise, are awarded 1 'cost' point.

The weight assigned the cost component should be increased by at least 1 point for treatments that cause discomfort, place the patient at significant risk for harm, or are inordinately time-consuming or inconvenient for either the patient or the caregiver.

According to this scheme, a given therapy or technique could be regarded as cost-effective if its benefit-to-cost ratio (a sort of 'how-much-bang-for-the-buck' index) is more than 1. Therapies whose cost exceeds their benefit (index is less than 1) are not considered cost-effective according to this system.

Having stated what I consider to be the most important current issues in home respiratory care, and having also provided a means for evaluating the various modalities discussed, I now summarize what I consider to have been the take-home message for each general area of home respiratory care

discussed during the conference. The four areas are long-term oxygen therapy (LTOT), long-term mechanical ventilation, airway clearance techniques (ACTs), and exercise.

Long-Term Oxygen Therapy

From this conference, conclusions can be drawn with respect to four different aspects of LTOT. First, oxygen-conserving devices work. They conserve oxygen. However, both patient selection and choice of device need to be individualized. No one device is best for every patient, depending on the disease, the breathing pattern, the level of physical activity, and so forth. And, we have to face the fact that with all of these devices, and to varying degrees among them, patient acceptance is not very good. Any device that is going to be used by only one half to two thirds of patients is not going to be cost-effective if we have to supply it to all of them.

Second, transtracheal oxygen (TTO) also works. It improves adherence (a new term for my working vocabulary from this conference). It increases the hours of use, and, from the results of the Nocturnal Oxygen Therapy Trial (NOTT),[16] there is good reason to assume that increasing actual oxygen use to 24 hours a day can improve survival beyond that in the "continuous oxygen" group in that trial. Several physiologic alterations also occur with transtracheal oxygen, affecting minute ventilation, breathing pattern, and, perhaps, dyspnea, among others.

The bad news is that relatively few patients are appropriate for transtracheal oxygen therapy. We have to face up to that fact, which even the staunchest proponents of this therapy will do. I have heard it said that TTO is appropriate for not more than 30% of patients, and the proportion may be far smaller than that. No doubt this varies with the clinician's practice setting, but transtracheal oxygen therapy is clearly not for most patients now considered candidates for LTOT. Mucus balls remain a serious problem, but are manageable with routines that have been worked out.

Nocturnal desaturation, in individuals who are not hypoxemic during the day, seems to identify a subgroup of COPD patients who are on a different prognostic or natural history track from their counterparts who sleep through the night without significant desaturation. However, the evidence is unconvincing that LTOT helps in these patients, and this is an enormously important matter in view of the Health Care Finance Administration (HCFA) regulations that permit reimbursement for this therapy for COPD patients who desaturate only at night.

Evaluation of lung disease patients for air travel is a controversial area. Patients who already meet criteria for LTOT, at whatever altitude they may live, should obviously receive oxygen during air travel. However, I question the recommendation that supplemental oxygen be provided during flight for clinically stable patients who are not hypoxemic at home but in whom it can be predicted that arterial P_{O_2} is likely to fall below 50 torr at altitude. Nobody really knows how clinically important such in-flight hypoxemia is. One thing we do know is that hypoxemia-related adverse events during commercial air travel are really rare—not just uncommon, but rare—and tend to occur in people who have recently been hospitalized or otherwise been treated for acute illness. How many clinically stable patients would have to be screened in order to prevent one of these rare occurrences, and would the provision of supplemental oxygen during the flight really prevent it? How many people should be supplied with oxygen on commercial flights? Twice as many as today? Ten times as many? This is an area that clearly needs further study in view of the costs and hassles currently involved in providing supplemental oxygen for patients during travel.

Long-Term Ventilatory Assistance

Now let us go on to long-term mechanical ventilation. We spent most of our time at the conference on this topic, appropriately so because it is an expensive modality and a lot of work is being done in this area, even though the number of patients using home ventilators is still quite small—perhaps not more than 1 to 1.5% of the number of patients on LTOT in the United States. General consensus of the conference participants was reached on 9 different aspects of long-term mechanical ventilation, as listed in Table 3.

Long-Term Oxygen Therapy vs Long-Term Ventilatory Assistance

One principle to emerge during the conference is that the best treatment for chronic hypoxemia is not

Table 3. Consensus of Points on Long-Term Ventilatory Assistance Attained during 1993 Journal Conference

1. The best treatment for chronic hypoxemia is not necessarily long-term oxygen therapy (see Table 4).

2. In appropriate patients, noninvasive ventilatory assistance can be effective in three separate clinical settings:
   - in acute respiratory failure (to avoid intubation);
   - as a transition phase in weaning from ventilatory support; and
   - for elective part-time ventilatory assistance in chronic ventilatory insufficiency.*

3. Non-invasive ventilatory assistance is preferable to tracheostomy for long-term ventilatory support.†

4. The initial choice for noninvasive ventilatory assistance should be nasal intermittent positive-pressure ventilation.†

5. Not every unweanable patient is a candidate for long-term ventilatory assistance.

6. The primary purpose for sending a ventilator-assisted patient home should be to improve that patient's quality of life, not to get him or her out of the intensive care unit or acute-care facility.

7. Large differences currently exist in patient selection and other important aspects of long-term ventilatory assistance in the United States.

8. A wide range of airway access devices, ventilators, and ventilator-function options is currently available for long-term ventilatory assistance.
   - Each has its place in appropriate patients and circumstances.
   - Clinicians and patients need access to appropriate expertise for apparatus selection and operation.

9. Infants requiring long-term ventilatory assistance pose special problems.
   - Less experience and expertise exist in the community than with adults.
   - Clinicians and patients need access to this expertise.

*In carefully selected circumstances, noninvasive ventilation may be appropriate for full-time ventilatory support; see text.
†With a few important exceptions, see text.

necessarily long-term oxygen therapy. For patients in several well-defined diagnostic groups, in whom hypoxemia is associated with chronic hypoventilation, ventilatory assistance may be more appropriate than supplemental oxygen. Table 4 summarizes this issue. Long-term ventilatory assistance (LTVA) means not only positive-pressure ventilation but also, in appropriate settings, therapies such as CPAP and bi-level positive airway pressure, as with the BiPAP™ device.

Long-term oxygen therapy is clearly indicated for COPD patients with resting hypoxemia; we all know the criteria, and they have not been improved upon. Time has shown the designers of the NOTT study[15] to have been pretty smart, and I think we can live with their entry criteria, which have been adopted by nearly all clinicians and healthcare systems for patient selection.

Patients who have COPD and hypoxemia with exercise, particularly when it can be shown that with supplemental oxygen they can do more and improve their quality of life, should also receive LTOT for that purpose. I think nobody would argue with that, although there are relatively few patients in this category, at least in terms of the number who actually wind up getting oxygen restricted to that circumstance. Hypoxemia during long-term mechanical ventilation is a sort of special case, but those ventilator patients at home who also are hypoxemic clearly need to have oxygen therapy.

Under oxygen therapy, in the "uncertain indication" category, goes the COPD patient with an arterial P_{O_2} of 60 torr or more while awake who experiences important nocturnal saturation. In my opinion, the category should be "doubtful" rather than "uncertain" for such patients, but there was definite consensus that studies establishing the long-term benefits of LTOT in this setting are lacking.

For chronic hypoxemia in patients with cardiopulmonary diseases other than COPD, such as interstitial fibrosis, cystic fibrosis, and congestive heart failure, we have a good rationale but no data. There are no acceptable studies in the three conditions I just mentioned, in sleep apnea syndrome, or in anything else—that is, acceptable studies in the sense that the NOTT[16] and Medical Research Council (MRC)[17] studies were rigorously designed, multicenter, prospective, controlled trials. We have to infer from the results of those studies, and that is a fairly important inference. There are not all that many chronically hypoxemic cystic fibrosis patients, but there are a lot of heart failure patients out there, and potentially a huge number with sleep apnea. For now, it is simply unknown whether LTOT would benefit them.

Table 4. Therapy for Chronic Hypoxemia: Oxygen or Ventilatory Assistance?

Status	Long-Term Oxygen Therapy	Long-Term Ventilatory Assistance
Definitely indicated	COPD with resting hypoxemia while awake COPD with hypoxemia during exercise Hypoxemia during LTVA*	Chronic ventilatory insufficiency in: neuromuscular disease central nervous system disease musculoskeletal/chest wall conditions Overt obstructive sleep apnea syndrome
Indication uncertain or doubtful	Nocturnal desaturation in COPD patients with P_{aO_2} > 60 torr while awake Hypoxemia in cardiopulmonary diseases other than COPD COPD with P_{aO_2} 60-70 torr	COPD; other obstructive lung diseases Amyotrophic lateral sclerosis Neuromuscular disease without hypercapnia Obstructive sleep apnea without symptoms

*LTVA = long-term ventilatory assistance.

Leslie Hoffman pointed out in one of the discussions that some patients who are referred for transtracheal oxygen therapy never live long enough to get their catheter tract matured and become accustomed to the therapy. In such cases, it is unlikely that such therapy is very beneficial. Likewise, I think, many patients in the terminal phases of illness who are prescribed home oxygen therapy probably don't survive long enough to experience much benefit. I am not just talking about increased survival because that is not the primary goal of home care, and, obviously, therapy has to be individualized. However, for hypoxemic patients with disorders other than COPD, I would like to see some sort of evidence of improvement in quality of life, such as increased walk distance, for example, or improvement on a dyspnea scale or some such, before committing those patients to home oxygen therapy for the remainder of their lives.

A stable arterial P_{O_2} value between 60 and 70 torr in patients with COPD poses another problem, even if they do not desaturate at night. Some clinicians, particularly in Europe, believe that the MRC and NOTT entry criteria were too strict and that similar benefit might be achieved in patients with less severe hypoxemia. At present there are no outcome data to help us with this, although there is no question that making patients with P_{O_2} values in the 60s eligible for LTOT would render this already expensive therapy enormously more so.

With respect to ventilatory assistance, it is clear that patients with neuromuscular disease complicated by progressive ventilatory insufficiency are the best candidates for this therapy to be identified so far. When nocturnal ventilation is begun at the appropriate time, these individuals do exceedingly well, as Patrick Leger and his colleagues,[11] among others, have demonstrated. Likewise, patients with central nervous system disease such as Ondine's curse and some others are also good candidates. Musculoskeletal diseases, and the thoracic cage disorders such as kyphoscoliosis, are perhaps a little farther down the list, but definitely in the "indicated" category.

To complete the "definitely indicated" list in Table 4, patients with full-blown sleep apnea syndrome need some kind of nocturnal ventilatory assistance, be it CPAP, bi-level positive airway pressure, or something else, with or without supplemental oxygen. In the lower category in the table, it remains pretty problematic whether LTVA should be routinely recommended for patients with end-stage COPD or amyotrophic lateral sclerosis (ALS). There are well-documented exceptions, especially in ALS—individuals in whom great improvement in quality of life has been achieved—but I suspect that the number of patients whose quality of life has not been improved, and has actually been rendered worse by attempts at home ventilatory support, far exceeds that number who have benefited. The advent of noninvasive forms of ventilatory assistance, such as nasal intermittent positive-pressure ventilation (NIPPV), makes it possible for selected patients to 'give it a try' without becoming committed to a tracheostomy, and this is an important advance.

Neuromuscular disease, such as Duchenne muscular dystrophy that is in a stable state without hypercapnia and acute respiratory failure, has not yet been added to the list of recommended indications.

Clinical Settings for Long-Term Ventilatory Assistance

Noninvasive ventilatory assistance as a general category of therapy has been discussed here in three distinct settings (see Table 3), and it is important to keep those distinct settings separate from one another. The database upon which practice rests in each case is different and should not be extrapolated across the different categories. Acute respiratory failure, with the idea of avoiding intubation and all of its inherent complications, is one setting. Another is the use of noninvasive ventilatory assistance (NIVA) as a transitional step during weaning from intubation and mechanical ventilation after acute respiratory failure. And the third is elective, part-time ventilatory assistance, whether this be CPAP, NIPPV, or some other form. As the experience of John Bach and his co-workers clearly demonstrates,[11] it is also possible in a few instances to progress from part-time, quasi-elective NIPPV to total ventilatory support 24 hours/day.

Noninvasive Ventilatory Assistance vs Tracheostomy

For most patients requiring long-term ventilatory assistance, the consensus view of the participants in this conference is that noninvasive techniques are preferable to tracheostomy. There are a few important exceptions, but very few in this case having to do mainly with secretion management in patients with poor glottic function or unusually copious, viscid sputum. But we can make the statement that noninvasive ventilatory techniques are preferable and should generally be attempted before tracheostomy.

Which Approach to Noninvasive Ventilatory Assistance?

The initial choice for noninvasive ventilatory assistance should be IPPB rather than negative pressure ventilation, pneumobelt, or some other possible approach. Once again there are exceptions, but these are very few, as described by Nicholas Hill in his review.[13]

Which Airway Access Device?

This conference has convinced me that the nasal mask should be the initial route to LTVA. That is a pragmatic thing, at least in this country, as it is the only airway access device generally available to the clinician. As is the case with oxygen conservation devices, a number of nasal masks are available, and no single one of them is best for all patients. It is definitely in the patient's best interests for the clinician to be familiar with the different brands and sizes available and to have several available to try, rather than attempting to apply some standardized or cookbook routine to all patients.

Thus, long-term ventilatory support should be noninvasive rather than via tracheostomy, positive- rather than negative pressure, and nasal mask rather than mouthpiece or full face mask. We must acknowledge the existence of specific clinical circumstances and of virtuoso-type expertise in a very few centers, in the presence of which other approaches may be appropriate, but for the great majority of clinicians (and their patients) we recommend the plan described here.

Should Unweanability Automatically Mean Long-Term Ventilatory Assistance?

The experts gathered for this conference have an aggregate clinical experience with well over 1,000 home ventilator patients. These experts are also in clear agreement, from managing or consulting on many others, that not everyone 'stuck' on a ventilator is a candidate for long-term mechanical ventilation. This last statement seems obvious, and no one could argue rationally against it, yet I cannot overstate how important it is or how often it fails to be appreciated in acute-care hospitals. Some patients never reach a state of medical stability that would make management outside the intensive care unit feasible. Others could be supportable in a less technology- and care-intensive setting but fail to meet the criteria of primary and secondary diagnoses, psychosocial resources, and other determinants of success in home care that Mary Gilmartin discusses in her article from this conference.[15]

Long-term home ventilatory support can be extremely successful, and associated with meaningful and enjoyable life for both patients and caregivers. However, its cost-effective application is highly dependent on patient selection—which often means *not* considering patients to be candidates for this therapy.

Why Send the Patient Home?

When ventilatory support at home is appropriate, the main reason should be to improve the quality of the patient's life rather than simply to get him or her out of the intensive care unit. Again, no one would disagree with that as a general principle. However, as someone who works in an acute-care hospital rather than in home care, my experience is that most patients are moved out of intensive care units to other locations for the primary purpose of reducing the expense of caring for them. We cannot ignore the importance of the money factor in this age, but we must keep in mind that as clinicians we are devoted primarily to the patient and that quality of life has to be number one on the over-all priority list.

Variations in Long-Term Ventilatory Assistance in Current Practice

Large differences in practice exist just in the United States, let alone in the other countries of the world. In many areas, the idea of NIVA has not yet arrived. We have heard at this conference that in muscular dystrophy, long-term ventilation is offered to patients and families in some regions of the country and not in others. Given the favorable results and extended survival reported here and elsewhere with LTVA in patients with neuromuscular diseases, such regional practice variations are hard to justify. It is not helpful to have a life-sustaining therapy at hand if people don't know about it or avail themselves of it. We need more data on the dimensions of this problem, but there is a clear need for improved communication and better education of both clinicians and patients.

Importance of Multiple Technical Options in Long-Term Ventilatory Assistance

A wide spectrum of devices and techniques are available for LTVA, and each has its place. Clinicians managing ventilator-assisted patients need access to the expertise of those specialists in this field who know how to select among them and best match the support to the patient's needs. Both pressure and volume ventilators have a place in long-term mechanical ventilation. I have been impressed with the papers that John Bach and his colleagues have written about the spectrum of support options, particularly with respect to the airway interface and how there is no single cookbook approach that will match the therapy to the patient's needs.[18-21]

Another way to put that is if we are not considering all the available options, we are not giving ideal therapy. Our patients are getting suboptimal care if we are not availing ourselves of the full spectrum of devices and expertise available. Obviously, there are logistic problems with that, in that not all patients can be cared for by John Bach or the other conference attendees. However, as an expression of consensus from this conference, it is important to point out that there is a need for access to this kind of expertise.

Special Problems Posed by Infants Requiring Long-Term Ventilatory Assistance

Infants who require ventilatory support in the home pose special problems for a number of reasons. Among other things discussed by Bob Kacmarek in his review,[14] these involve the ventilator, the airway access, and the growth of the patient during the course of management. There are less experience and expertise out in the clinical world in the area of small children and infants than with older children and adults. Again, the need to access this expertise is very important, even though the numbers of patients are relatively small.

Exercise

Physical Reconditioning

The summary statements that can be made here are not very controversial, at least today. All patients who are physically capable of exercise can benefit from it. We know that the benefit increases with the intensity of the exercise. We are cautioned, though, that it needs to be an appropriate kind of exercise, and that training for the wrong activity is not going to improve the patient's quality of life. Physical capacity developed through training falls off with neglect, and thus the exercise regimen needs to be sustained for the life of the patient if the benefit is to be retained. Adherence is a problem. Adherence to exercise faces the same problems in patients as it does in the general population, who are well

aware that they should stop smoking, use seat belts, eat better, and exercise more, yet usually do not follow through with these things. Those problems are the same in our patients.

Breathing Training

My second conclusion with respect to exercise is that breathing training helps. Breathing training fell into a kind of stagnation for many years. Both old data and newer work support its efficacy, but clearly there are many quality-of-life-type benefits to be attained by training our patients to voluntarily breathe in patterns that decrease dyspnea and create more favorable physiology.

Ventilatory Muscle Training

The conclusion here is that ventilatory muscle training is unpleasant and doesn't get you much. I think we can probably lay this one to rest. Most of us have long since stopped trying to get our patients to engage in training regimens, if we ever did, despite the initially encouraging reports and more recent modest evidence for efficacy.[10]

Airway Clearance Techniques

This subject is rife with terms and acronyms unfamiliar to many clinicians, all of which are defined and explained in the review by Karen Hardy.[8] My only general observation or concluding remark about this aspect of home respiratory care is that, despite a fair number of published studies, the state of the art (or, rather, state of the science) is still pretty inadequate. Much of the literature is essentially anecdotal or nonquantitative, and practice is largely determined by individual clinician experience and bias. This is not because the people working in the area are not smart or don't know the scientific method. Instead, it is because the system they must work with and the ways of quantitating it, are notoriously hard to study. This is especially true for COPD, and essentially applies to all disorders other than cystic fibrosis. Airway clearance techniques relate very much to quality of life, and illustrate the need to use some measure of that in attempts to determine efficacy, particularly because some of the techniques used are intrusive, uncomfortable, and demanding of the patient's effort.

Cost-Benefit Assessment of Home Respiratory Care Modalities Discussed at the Conference

In Tables 5-8, I have attempted to assess each of the therapies we have discussed in the context of cost-benefit and quality of life. Admittedly this assessment is arbitrary and largely subjective, but I feel the exercise is worthwhile nonetheless in view of the stakes involved—both for the healthcare system and for our patients.

Oxygen Therapy (Table 5)

Although it is fairly expensive, LTOT for stable patients with resting hypoxemia rates high. It increases survival and improves quality of life by several measures. In the case of hypoxemia only during exercise, it is a little less convincing but still well into the benefit side of the equation. For nocturnal hypoxemia, and for modest hypoxemia that falls short of the NOTT and MRC criteria, however, the story is different. Although the rationale in each case is clear, a lack of experimental data and the huge numbers of patients involved should keep us from rushing to supply these people with LTOT.

Oxygen-conserving devices are hard to evaluate because so much depends on whether patients use them. When a motivated patient experiences increased mobility and the ability to be away from home for many hours at a time, the benefit is obvious. On the other hand, the routine use of these devices cannot be recommended because such patients are unusual. Transtracheal oxygen is cost-effective for those relatively few patients who are highly motivated and capable of following the regimented protocol and can afford both the initial procedure and the ongoing expense of this delivery system.

A lack of relevant data hampers the meaningful assessment of whether patients who do not meet hypoxemia criteria for LTOT should be provided with oxygen during air travel. Given the expense and inconvenience to both patient and clinician, I am not eager to pursue this vigorously in my patients who are clinically stable. Of the medical mishaps that do occur during commercial air travel, many affect patients recently discharged from the hospital or otherwise not in a clinically stable state; if air travel becomes necessary for such individuals, an assessment for in-flight hypoxemia is probably a good idea.

Table 5. Assessment of Home Oxygen Therapy from a Cost-Benefit Perspective

Modality	Cost-Effective?*	Comments
LTOT in COPD with resting hypoxemia	Yes (5/3)	Increases survival; decreases hospitalizations; improves exercise, sleep, appetite, sexuality, neuropsychological function
LTOT in COPD with hypoxemia occuring only during exercise	Yes (4/3)	Improves exercise capacity
LTOT in COPD with hypoxemia occurring only during sleep	No (1/3)	No documented efficacy in this subgroup of patients
LTOT in COPD with P_{aO_2} 60-70 torr	No (1/3)	NOTT and MRC entry criteria may have been too strict, but no data
Oxygen-conserving devices	Varies	Improve oxygenation in some patients; conserve oxygen; extend range of portable units; reimbursement a problem; poor patient acceptance
Transtracheal oxygen	Varies	Increases hours of use; may decrease dyspnea; better oxygenation in a few patients; expensive; only a few patients appropriate
Oxygen during air travel for patients who meet criteria for LTOT at rest at home	??	Recommended on basis of predictable worsening of known hypoxemia
Oxygen during air travel for patients who do not meet criteria for LTOT at rest at home	No (1/3)	Clinical risk to patient unknown, even with predicted $P_{aO_2} < 50$ torr during flight; large hassle factor and expense

*Number in parentheses is benefit-cost ratio from Table 2 (in appropriately selected patients). If ratio is < 1, the modality is not cost-effective.

Ventilatory Assistance (Table 6)

In patients with overt sleep apnea syndrome who actually use the device long-term, CPAP or bi-level positive airway pressure can substantially improve quality of life, and the cost is modest, especially in view of the paucity of acceptable therapeutic alternatives.

In my opinion, the cost-benefit/quality-of-life assessment is pretty unfavorable for patients who require long-term ventilatory support and cannot be started down the track in Figure 2 that leads to home care. Diagnosis also has a very important impact in LTVA, with the cost-benefit relationship being favorable for many patients suffering from neuromuscular, neurologic, or musculoskeletal disorders, but unfavorable for most individuals with end-stage COPD or ALS.

I am convinced that, if it can be accomplished, NIVA is decidedly superior to the use of a tracheostomy, and the cost-benefit analysis bears this out. Although I have not mentioned it previously, phrenic nerve pacing (electrophrenic respiration) is often discussed when patients with phrenic nerve lesions or high spinal cord injury are considered for LTVA. The participants were unanimous in their lack of enthusiasm for this technique, which is very expensive and seldom successful long-term, at least in the collective experience at this conference.

Frog breathing (glossopharyngeal respiration), on the other hand, costs nothing and is used successfully by many patients who are otherwise ventilator-dependent to achieve increased off-ventilator time, and with it substantially improved mobility and safety in the event of a power failure. Thus, this technique, once learned, is definitely cost-effective.

Exercise (Table 7)

In patients who are motivated to undergo the training programs and continue to use the techniques, both physical reconditioning (leg and arm exercise) and breathing training are clearly cost-effective. They can enable patients to do more and

Table 6. Assessment of Long-Term Ventilatory Assistance from a Cost-Benefit Perspective

Modality	Cost-Effective?*	Comments
CPAP or bi-level positive airway pressure in obstructive sleep apnea syndrome	Yes (4/3)	Variably reverses clinical manifestations of sleep apnea; variable patient acceptance
Long-term ventilation in acute-care facility	No (1/5 or 2/5)	Most expensive long-term care setting; severe disruption of neuropsychiatric and psychosocial function
Long-term ventilation in long-term acute care facility or ventilator unit	No (3/5)	Less expensive than acute-care hospital; reduction in impact on quality of life vs that in acute-cate hospital uncertain
LTVA in patients with neuromuscular, CNS, or musculoskeletal disorders	Yes (4/3 or 5/4)	Survival almost certainly extended; hospitalization needs reduced; improved psychosocial function
LTVA in patients with COPD or ALS	No (3/4 or 4/5)	Cost often more than with neuromuscular or musculoskeletal patients because of older age, unstable disease, other medical problems
Noninvasive ventilation (as compared to tracheostomy) for LTVA	Yes (4/3 or 5/4)	Preferred by patients; improved communication; fewer infections (not for patients with secretion problems)
Phrenic nerve pacing	No (2/5 or 3/5)	Very expensive; infrequent long-term success; poor patient acceptance
Frog breathing	Yes (4/1)	Once learned, increases patient's mobility and ventilator-free time

*Number in parentheses is benefit-cost ratio from Table 2 (in appropriately selected patients). If ratio is < 1, the modality is not cost-effective.

to control severe episodes of dyspnea. Ventilatory muscle exercise, on the other hand, rates low on my cost-benefit scale. It is unpleasant and whether it really does anything clinically significant is pretty much in doubt. At present the same goes for ventilatory muscle training to aid in weaning from prolonged mechanical ventilation in the intensive care unit.

Airway Clearance Techniques (Table 8)

Here the key to cost-effectiveness and improved quality of life is patient selection. Postural drainage, percussion, and vibration are labor-intensive and can be uncomfortable for the patient, but when effective can decrease cough and sputum expectoration during the day and can hence improve quality

Table 7. Assessment of Home Care Modalities Related to Exercise from a Cost-Benefit Perspective

Modality	Cost-Effective?*	Comments
Physical reconditioning	Yes (4/1 or 4/2)	Improves exercise capacity, appetite, and sense of well-being; requires continued use for sustained benefit; many patients cannot or will not adhere to regimen
Breathing training	Yes (4/1)	Helps to control dyspnea; must be consciously performed in order to be effective
Ventilatory muscle exercise	No (1/1 or 1/2)	Unpleasant; patient acceptance poor
Ventilatory muscle training as an aid to ventilator weaning	No (1/2 or 2/3)	Labor-intensive; unpleasant for patient; lack of convincing evidence of efficacy

*Number in parentheses is benefit-cost ratio from Table 2 (in appropriately selected patients). If ratio is < 1, the modality is not cost-effective.

Table 8. Assessment of Airway Clearance Techniques used at Home from a Cost-Benefit Perspective

Modality	Cost-Effective?*	Comments
Postural drainage, percussion, vibration	Varies	Labor-intensive; expensive if paid caregiver required; effective in some patients (eg, cystic fibrosis; some bronchiectasis patients)
Forced exhalatory technique Active cycle of breathing Positive expiratory pressure Autogenic drainage	Yes (3/1 or 4/2)	Helpful in selected patients; can be tedious, affecting adherence; benefit requires continuous use
High-frequency chest-wall compression ("the vest")	No (1/4 or 2/5)	Expensive; documentation of efficacy unconvincing

*Number in parentheses is benefit-cost ratio from Table 2 (in appropriately selected patients). If ratio is < 1, the modality is not cost-effective.

of life. The various breathing techniques for sputum removal (forced exhalatory technique, active cycle of breathing, and autogenic drainage) and the positive expiratory pressure (PEP) device are inexpensive and helpful for selected patients. However, high-frequency chest-wall compression seems a pretty expensive proposition in the face of currently available data on its efficacy.

Recommendations for Additional Studies and Other Actions

I conclude by mentioning several areas in which the availability of good data would be of major help to both clinicians and health economists. These areas 'stuck out' during the paper presentations and ensuing discussions, and I would like to be able to speak for the group in urging that studies in these areas be undertaken wherever and as soon as possible.

How many patients are there in the United States on LTOT? Ours is just about the only country for which this information is not available, and yet more patients have received this therapy in the United States than in all other countries of the world combined. There needs to be a National Home Oxygen Day or something, during which every patient treated with LTOT is somehow tallied, along with their main diagnoses, how much oxygen is prescribed and when, and what equipment is used to deliver it. The AARC and the profession in general enjoy good relations with physician groups and with the home care industry, and somehow it ought to be possible to get this information.

And while we are at it, how many home ventilator patients are there in this country? Attempts to estimate this number have been made, but hard data would be infinitely more valuable. How many patients are there today who use nasal CPAP or bi-level positive airway pressure for treatment of sleep apnea? What kinds of problems do they have with the treatment, and what would they like to see changed? How many infants and small children are managed on ventilators at home? Answers to these questions would assist health resource planners, third-party payors, equipment manufacturers, home care companies, clinicians, and patients alike.

A NOTT-type multicenter study has been called for to determine whether patients who desaturate only during sleep survive longer and/or function better if treated with LTOT. In contrast to the studies mentioned above, this one strikes me as a 'long run for a short slide.' It would require enormous numbers of patients and cost many millions of dollars if it could be done at all. I think such a study is unlikely to be undertaken.

What happens to COPD patients when they fly on commercial jetliners? I don't mean "What is the predicted P_{aO_2} at altitude based on a test done on the ground," but what is the actual saturation throughout the flight, and what happens to the patient's symptoms and vital signs, and what aspects of the patient's history and management and activities are most predictive of problems during the flight? Many patients with COPD experience marked desaturation every night in their own beds while they sleep—as do some apparently healthy people. Under what circumstances would similar desatura-

tion during air travel be harmful to them? It would seem like a good idea to answer these questions before recommending supplemental oxygen for every person whose P_{aO_2} is likely to fall to 50 torr during a commercial airline flight.

What studies should and could be done to advance our knowledge about LTVA? One might be the derivation and clinical validation of a scoring system to predict successful discharge and long-term, successful survival at home in unweanable patients following acute respiratory failure. Another might be a comprehensive survey of a national cohort of muscular dystrophy patients or other potential candidates for elective initiation of LTVA, to document differences in management in different areas of the country. If such a study confirmed the impression raised at this conference that life-sustaining measures are not even discussed with many patients or their families, its results could be used in designing nationwide educational efforts to provide all patients with equal access to, or at least awareness of, currently available therapies.

Good studies are clearly needed on the effectiveness of the various airway clearance techniques, but I don't know what form they should take or what should be studied. This is a very difficult area, and definitive data are unlikely to burst onto the scene any time soon.

Finally, there is a great need now for studies to document the value of the respiratory care practitioner, not just in home care but in every clinical arena. A lot of studies have been done by respiratory therapists on how devices work and how best to treat disease. But a study done by a respiratory care practitioner on how to treat disease or how to work up a patient does not help the respiratory care profession itself very much. We need studies on the value of the respiratory care practitioner and of that person's unique expertise as consultant, educator, or coordinator of patient care. Unless we can document that kind of value, objectively and with appropriate study design, there is an increasing uncertainty to the profession's future.

Concluding Comment

This conference brought together an impressive group of clinicians, investigators, teachers, and administrators working in home respiratory care. In 2 1/2 days of discussion the participants articulated the state of the art in the most important areas of this important and growing field of healthcare. This articulation took place not just from the podium, but also during the discussions following each formal presentation, and in less formal interactions among the participants.

In the course of the conference, and with the publication of these two special issues of RESPIRATORY CARE, the objectives of the Journal Conference Series have been realized. These objectives are to identify a clinically important aspect of respiratory care in which rapid progress is occurring but on which the existing literature is incomplete; to bring together a critical mass of the profession's leaders, both seasoned veterans of these conferences and newcomers to the process; to address squarely those facets of the topic at hand with which the readers of the Journal are most directly concerned; and to create a unique and valuable document that helps to increase our knowledge, expand our skills, and improve the care of our patients. It has been a challenge and a privilege to summarize this process in this article, and to introduce the excellent, authoritative papers that follow it.

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Demographics and Financial Impact of Home Respiratory Care

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Prologue

The term "home healthcare" encompasses an array of services and products furnished by distinct and separate providers. Over the years, reimbursement practices have contributed to the segmenting of the home healthcare system into defined and separate entities. Although not a directly reimbursable service, respiratory home care has nonetheless become a widely utilized resource. Healthcare reform, coupled with compelling demographic and epidemiologic imperatives, creates an even greater need for high quality respiratory home care services in the years ahead. To that end, timeworn and outdated reimbursement guidelines and the associated mindsets must be modernized. The perpetuation of yesterday's thinking only delays further the realization of tomorrow's promise with respect to respiratory home care services.

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Background

The provision of medical care in the home setting has a long and rich history in the United States. The vibrant and ever-expanding system of home healthcare services we see today actually traces its origins to 1893 and the vision of two nurses in New York, Lillian Wald and Mary Brewster.[1] Nurses Wald and Brewster, realizing that medical care rendered in the

home could offer important advantages over care in institutional settings, invited other nurses and social workers to join them in the creation of the Henry Street Settlement and to begin providing health and social services in the home. Nurses and social workers were assigned to go into the community and visit patients who were otherwise unable to transport themselves to the hospital for care. The system expanded and, in a few years, became a major source of healthcare services for the indigent and the less fortunate of the populous. It was this concept of sending nurses and social workers to make home visits that evolved to become what is known today as the Visiting Nurses Association (VNA).

Throughout the first half of this century, home healthcare continued to be a resource provided primarily for the economically disadvantaged. Accordingly, VNAs were typically funded through a combination of public and philanthropic contributions and were viewed as an extension of public health services. However, during the same time period, several religious orders likewise developed programs to provide home visits to members of their congregations. Although the impetus for these home health programs was to render medical care, spiritual support and rejuvenation were provided as well. In 1947, the nation's first hospital-based home care department was established at Montefiore Hospital in New York City.[1] Dr EM Bluestone, director of the hospital from 1928 to 1950, believed that in addition to treating inpatients hospitals also had a certain social responsibility to care for its patients once they went beyond its doors. Dr Bluestone's vision and actions formally established a link between care provided in the hospital and care provided in the home.

Medicare Program

The home healthcare industry continued in the previously described vein without significant growth until 1966, at which time the federally funded Medicare Program was signed into law by President Lyndon Johnson. Designed to provide subsidized healthcare services to the nation's increasing elderly population, the Medicare Program contained two distinct provisions that had a significant impact on the growth of home healthcare. Medicare Part A, the hospital benefit, contained a mechanism to fully reimburse for intermittent skilled visits provided to homebound beneficiaries. Medicare Part B, the optional companion benefit to Part A paid for by the beneficiary, contained a mechanism to partially reimburse for items of medically necessary equipment and supplies provided for use in the home. The coupling of the Part A and Part B Medicare home healthcare benefits was viewed as a logical extension of the primary intent of the Medicare Program—to reduce the likelihood that our nation's elderly would be financially ruined if and when they required healthcare services.

The impact of the Medicare Program on the growth of home healthcare services cannot be underestimated. For the next 25 years (1966-1991), the number of home health providers arriving on the scene grew exponentially. And, it was not only the size of the Medicare-aged population that drove the expansion: Private insurance companies, looking for ways to manage alarming increases in the costs of traditional hospital care, began to recognize home healthcare as a viable, cost-effective strategy for rendering healthcare services. This imperative became especially compelling during the 1980s as the nation's total annual costs for healthcare skyrocketed, expanding each year at more than double the rate of inflation. So severe was the escalation that by the end of 1993 total healthcare expenditures were estimated to top $900 billion for the year, consuming about 14% of the gross domestic product.[2] It is little wonder then that healthcare reform has found its way onto the national agenda, and, in late 1993, President Clinton delivered a long-awaited reform package to Congress and to a skeptical public.

Costs of Home Healthcare

While the nation's overall healthcare system has reached a real and serious financial crisis, home healthcare truly represents one of the few bright spots. For example, in 1991, total expenditures for home healthcare amounted to $11.1 billion or approximately 2.5% of the total $850 billion spent for all healthcare services for the year.[3] This represented a modest annual increase of 8% from the $6.1 billion spent on home health in 1987. During the same period, the consumer price index (CPI) grew at 19.8%. By way of comparison, between 1987 and

1991, hospital costs increased 46% (more than twice the CPI) with physician and other medical costs increasing by 32% (almost twice the CPI). On the other hand, expenditures for home health grew at one half the CPI and one must wonder what the increases in both hospital and physician costs would have been without home healthcare.

Although the future of the U.S. healthcare delivery system remains unclear in terms of the President's reform initiative, the future of home healthcare, by all accounts, remains rosy. A recent forecast by Frost and Sullivan projects a total home healthcare market of $20 billion by 1996.[4] Moreover, the U.S. Department of Commerce recently identified home healthcare as the fastest growing segment of the healthcare industry.[5] Whatever shape our nation's healthcare delivery system ultimately takes, home healthcare is expected to be a highly visible strategy and an important complement to more traditional institutional-based care.

Defining Home Healthcare

Home healthcare is defined as "the provision of services and equipment to the patient in the home for the purposes of restoring and maintaining his or her maximal level of comfort, function, and health."[6] Home healthcare can be provided to individuals with acute illness, exacerbations of chronic illness, or long-term disability, who do not require hospitalization. Although traditionally provided following discharge from an institutional care setting, home healthcare is now increasingly being used as an alternative to such care. This trend is certain to continue as physicians become more comfortable with the notion of prescribing home healthcare.

While it is easy to offer a definition of home healthcare, it is somewhat more difficult to describe the types of home healthcare providers that exist. This difficulty exists because over the years, reimbursement strategies favored segmenting home health into discrete scopes of service. Thus, the type of care/service provided by a particular home health organization is determined in great measure by reimbursement policies, most notably those promulgated by the Health Care Financing Administration (HCFA). Five major types of home healthcare services are provided under the present system, each possessing its own unique reimbursement mechanism.

Home Health Agency

A home health agency is an entity that provides visits by skilled healthcare professionals on an intermittent basis. Third-party payors reimburse an agency 100% on a per-visit basis, which is expected to be inclusive of all direct costs associated with the visit (eg, salary, supplies, travel, and administrative overhead). Home health agencies must be licensed by their state regulatory boards and certified under Medicare Conditions of Participation if Medicare reimbursement is sought for visits to homebound beneficiaries. In addition to providing skilled nursing services, a home health agency can also arrange for intermittent visits by a member of one or more of the following allied health professions: physical therapy, occupational therapy, speech therapy, and medical social work. Regrettably, respiratory therapy is not one of the allied health professions recognized for per-visit reimbursement under the Medicare Part A program. However, in 1991, HCFA did begin to allow Medicare-certified home health agencies to hire respiratory therapists and include their salary and benefits on the annual cost report.[7] Still, under this cost allocation process, there is no per-visit reimbursement for home visits made by a respiratory therapist.

To be eligible for reimbursement for intermittent skilled home visits under Part A, the beneficiary must satisfy certain criteria. An initial nursing assessment is conducted to establish that the beneficiary is homebound and will benefit from a planned series of home visits to facilitate a resumption of activities of daily living. On average, 12 to 15 visits are provided over the course of 3 to 4 weeks. In certain instances, home health aides may be utilized to perform necessary services established in the treatment plan, which could include help in personal care, exercises, and walking. According to recent HCFA figures, approximately 7,163 of the 13,900 home health agencies nationwide are Medicare-certified.[8]

Some home health agencies also provide in-home hospice care to terminally ill Medicare recipients. Reimbursement for hospice care is on a capitated, per-diem basis and is inclusive of all costs as-

sociated with providing care. According to the National Association for Home Care, there are about 1,400 in-home hospices nationwide.[9]

Home Medical Equipment/Respiratory Therapy Provider

Sometimes referred to as durable medical equipment (DME) suppliers or vendors, home medical equipment/respiratory therapy (HME/RT) providers arrange for the selection, delivery, setup, use, and ongoing maintenance of medically necessary equipment in the home. In addition to furnishing prescribed equipment and supplies, HME/RT providers spend considerable effort and time teaching the patient and/or caregiver how to care for equipment properly, safely, and in full accordance with their current physician's prescription. HME/RT providers receive reimbursement from third-party payors for the sale or rental of the prescribed equipment—typically at about 80% of an established schedule. The remaining, unpaid portion, referred to as the co-payment, is the responsibility of the patient. For Medicare beneficiaries, the co-payment is usually covered through a Medicare supplemental insurance policy.

Respiratory equipment and supplies represent an important portion of the total volume of HME provided. Although some respiratory equipment is of modest complexity (eg, a standard compressor-nebulizer), some items are more complex (eg, an apnea monitor or mechanical ventilator). Thus, to ensure that each patient receiving respiratory equipment is properly trained to the degree required by the complexity of the equipment, HME/RT providers typically retain the services of qualified respiratory therapists. Home care respiratory therapists conduct initial and follow-up home visits to monitor equipment performance and usage in terms of the physician's intended expectations. At times and under a specific and individualized plan of treatment, home care therapists may also conduct ongoing clinical assessments, focusing on the patient's response to the prescribed therapy. Within the past few years, home care therapists have also become much more visible in the performance of in-home diagnostic testing for sleep disorders, a specialty that is certain to grow even further.

In light of the array of professional services provided by home care respiratory therapists, it is unfortunate that HME/RT providers do not receive direct reimbursement for such services. Because reimbursement rates reflect only equipment and supplies, home care respiratory therapists are considered a direct expense to HME/RT providers. Given the current climate of cost-containment, one must seriously question what impact further draconian slashes in reimbursement rates for respiratory equipment will have on the ability of HME/RT providers to keep qualified respiratory therapists on staff.

Home Infusion Therapy Provider

A third type of home healthcare service is provided by organizations that compound, mix, dispense, and arrange for the administration of pharmacologic agents and biologicals. Home infusion therapy providers utilize the services of two types of healthcare professionals—registered pharmacists and registered nurses with experience in parenteral administration techniques. The typical role of the pharmacist is to obtain and verify the prescription from the physician and to compound the materials according to the individual needs of each patient. Home nurses, in turn, work with the patient or caregiver to ensure proper and safe administration of the prescribed solutions or medications. Home infusion therapy nurses also gather assessment-based information on outcomes and complications and provide ongoing feedback to both the physician and pharmacist. The team approach has been found to be especially important because of the complexity of home infusion therapy. In fact, experience has shown that successful home infusion therapy providers have refined the team concept, whereas less-than-satisfactory providers can be characterized as having poorly coordinated and fragmented services.

Home infusion therapy providers are reimbursed for the medications and solutions. Sometimes a per-diem charge is also included to offset the expense of home nursing visits; however, it is not uncommon for charges for such visits to be included in the reimbursement for the product and supplies. Although reimbursement rates for home infusion therapy tend to be the highest in home healthcare, this therapy is still regarded as cost-effective, given the alternative of being treated in an acute care setting. In an effort to further maximize cost savings, some providers have established ambulatory infusion therapy sites,

thereby reducing the high expense of home nursing visits.

Homemaker Services & Private Duty Nursing

Homemaker services and private duty nursing are usually provided only on a private pay basis. Homemaker services are provided on a shift basis, typically 4 hours, to individuals who are homebound and otherwise unable to fully care for themselves. Homemaker services include assistance with personal hygiene, meal preparation, and other activities of daily living. Private duty nursing is also provided on a shift basis (typically 8 or more hours), and may cover an entire 24-hour period. Full-time home nursing care is provided to patients who are bed-bound and receiving one or more 'high-tech' medical interventions, for example infusion therapy or mechanical ventilation. Services therefore include all regular skilled nursing functions such as medication administration, wound care, ventilator care, patient assessment, or any other treatments prescribed by a physician. At times, home nurses may use medical equipment and supplies furnished by an HME/RT provider. Although not a Medicare benefit, many commercial payors are beginning to reimburse for full-time home nursing in situations in which costly institutional care is the only alternative. Whether providing in-home hospital care represents a growing trend, remains to be seen.

Regardless of the exact type of home healthcare service one considers, all share a common link—the delivery of healthcare services in the least costly site, the patient/caregivers' home. Although some may still question what the true impact and potential of home healthcare is (or will be!) in terms of quality, cost-effectiveness, and outcome, sentiment is growing for ever greater use in the future. The architects of healthcare reform will, no doubt, look long and hard at the possibilities. In the specific area of home respiratory care, the evidence is already compelling.

Forces Favoring Growth in Home Respiratory Care

Demographics

During the early part of the 21st century the nation will witness an unprecedented growth in the number of older Americans as the baby-boom generation continues to age. The older population—persons 65 years or older—numbered 31 million in 1989 and represented 12.4% of the U.S. population.[10] This population increased 18% from 1980 to 1989, compared to a 7% increase for the under-65 population for the same period. By the year 2050, the older population is expected to double, to almost 69 million. Of greater significance, the number of Americans age 85 or older increased 165% between 1960 and 1980 and is expected to be 7 times larger by 2050.

Fueling the growth of the older American population is an increase in the life expectancy. For example, U.S. Bureau of Census statistics reveal that the life expectancy for Americans increased from 69.7 years in 1960 to 74.9 years in 1989.[11] By the year 2010, it is estimated that the life expectancy for Americans will be 77.9 years.

The growing number of older Americans will be accompanied by an increase in the number of chronic health conditions. This is sure to further strain the nation's already beleaguered healthcare delivery system, in terms of both utilization and cost. As one would expect, older patients tend to require more healthcare services than do younger patients. And, although not all older patients experience chronic pulmonary disease as a primary condition, pulmonary complications often do constitute a secondary diagnosis. Fortunately, much of the healthcare needed by older patients can be safely rendered in the home, a fact that will undoubtedly increase the demand and need for home care services.

Epidemiologic Considerations

The incidence of chronic respiratory disease in our society is an established fact. According to the Centers for Disease Control, chronic obstructive pulmonary disease (COPD) and related conditions have been the fifth leading cause of death since the late 1970s.[12] It is now conservatively estimated that as many as 1,000,000 patients may be afflicted with COPD. Chronic asthma, now estimated to afflict 12 million individuals, increased by 29% from 1980 to 1987.[13] The National Commission on Sleep Disorders Research recently suggested that as many as 40 million Americans may be chronically ill with various sleep disorders. An additional 20 to 30 mil-

lion more experience intermittent sleep-related problems.[14] The number of individuals diagnosed with HIV/AIDS is expected to exceed 1.5 million by 1997.[15] It is therefore clear that the patient population requiring acute and chronic respiratory care is growing at a significant rate. It is likewise evident that such care cannot be provided totally in the traditional acute hospital setting. Rather, alternate treatment strategies, such as home care, will be utilized, reflecting an efficacious, cost-effective collaboration between the patient, physician, and home care provider.

Cost-Effectiveness of Home Healthcare

The evidence of the cost-effectiveness of home healthcare is steadily becoming more attractive, in light of healthcare reform. This is especially true for patients afflicted with chronic respiratory problems. In the early 1980s, the then U.S. Department of Health, Education, and Welfare (HEW) sponsored a study that followed 775 COPD patients who received home respiratory care from qualified respiratory therapists. The results of the study showed readmissions to the hospital dropped from 1.28/year before home care to 0.55/year following home care. Furthermore, for those patients who were re-admitted, the length of stay decreased from 18.2 to 5.7 days. The total savings, in 1980 dollars, was estimated to be $1,097,250.[16] In 1982, under the direction of former Surgeon General C Everett Koop, a pilot program in Maryland provided home respiratory care to ventilator-dependent children and compared hospital costs to home care costs. It was determined that home care resulted in savings of more than $15,000/patient/month. Over the 34-month period of the program, $3.1 million in savings were realized.[17] In 1991, a similar study by the State of Illinois of ventilator-dependent infants receiving respiratory home care versus traditional hospital-based care, saved the State more than $4 million during a 48-month period.[18]

In 1990, a Gallup survey studied the cost of providing hospital care to chronic ventilator-dependent patients. The survey estimated that there were more than 11,500 such patients in U.S. hospitals, costing an estimated $789/patient/day, totaling more than $9 million/day. Efforts to discharge stable and otherwise suitable ventilator-dependent patients, hampered by outdated reimbursement policies, took an average of 35 days. Such delays translated to an excess of $27,000/patient in unnecessary hospital costs.[19] It was clear that the tremendous cost-savings possible through the use of respiratory home care was not yet fully realized by payors at all levels, governmental and private alike.

Finally, in 1991, an economic analysis by Lewin/ICF focused on the effects of the availability of home care services on the overall cost of care for patients in three separate diagnostic categories, one of which was COPD.[20] It was determined that $520/patient/episode could be saved if a COPD patient received home care instead of only hospital-based care. With an estimated population of more than 93,000 new COPD patients/year, annual savings would amount to more than $48 million. Given the prevalence of COPD in our society, these are indeed staggering figures.

The current climate of healthcare reform is certain to focus attention on cost-effective and efficacious strategies to provide quality care. Respiratory home care is increasingly being recognized as a viable intervention with a significant cost-savings impact. It is clearly time for archaic and outmoded reimbursement policies to be replaced with more realistic guidelines so that access to quality respiratory home care can be more universally realized by the millions of Americans in need of such care. To delay in doing so will only translate to more healthcare dollars being inappropriately and unnecessarily wasted.

The Future of Home Respiratory Care

As the nation continues its quest to bring escalating healthcare expenditure under control, there is no question that we will witness significant changes in the tradition-bound thinking that has contributed to a system viewed by many to be out of control. Simply stated, the acute care hospital can no longer be viewed as the primary site for healthcare services. Rather, providers, payors, regulators, and patients alike will be challenged to adopt newer approaches to streamline the cumbersome and wasteful strategies of years past. Novel concepts such as managed care, transitional care, outcome-based reimbursement, provider networks, and the delivery of seamless healthcare are already in evidence na-

tionwide. An especially refreshing paradigm is the "healthcare continuum."

Healthcare Continuum

The notion that there are several sites where healthcare can be delivered is certainly not new. What is new, however, is an intended and coordinated effort to determine which site (or sites) is most appropriate for a given patient presenting with a given medical condition. Figure 1 depicts the various venues that comprise the healthcare continuum. The acute care hospital, which represents one end of the continuum, is the most regulated and controlled site, and, not surprisingly, is also the most expensive. By contrast, home healthcare represents the opposite end of the continuum; because it is the least regulated and controlled, home healthcare is also the least expensive site for the delivery of healthcare. As one moves across the continuum from the acute care hospital toward home healthcare, the cost of providing care is reduced. Ideally, patients can move from one care setting to another as the acuteness of their illness and dependence on more expensive technology is reduced. An important facet of this approach to providing healthcare services is ensuring that all relevant information about treatment plans, medical interventions, and assessment of outcomes accompanies the patient from site to site. This enhances the likelihood of continued cooperation between the various providers and reduces the incidence of duplicated care. As the patient moves along the continuum, the prescribing physician must be kept informed of the patient's progress in attaining pre-established goals and readiness for referral to the next appropriate venue of care. Active and deliberate movement of patients along the healthcare continuum can reduce the occurrence of prolonged and costly stays in institutional settings when less costly alternatives can be readily accessed. Healthcare policymakers and payors at all levels increasingly are turning their attention to the concept of transitional care. Home healthcare is certain to be viewed in a favorable light when placed in this context.

Optimizing Home Care Outcomes

The growing acceptance of home healthcare as a viable venue in the healthcare continuum places new pressures on home care providers. Skeptics, bound by more traditional conventions, will continue to question whether successful medical outcomes can be consistently attained in the relatively uncontrolled home environment. Such arguments are further strengthened by the reality that, often, home healthcare relies on medically naive caregivers (eg, the patient, family, or significant others) who may be required to provide care that, at times, may be highly technical in nature. Fortunately, numerous success stories aptly demonstrate that, in fact, with proper education, training, and follow-up, medically naive caregivers can collaborate with home healthcare providers to achieve successful outcomes.

Central to the attainment of successful home care outcomes on a consistent basis is for the home healthcare provider to approach each new referral in an organized manner. As part of the admission process, an individualized assessment is conducted to identify specific needs or problems requiring attention. Identified needs or problems are in turn used to formulate one or more goals or objectives, which if realized will satisfactorily mitigate the presenting needs or problems. Finally, specific actions or interventions are planned and implemented to facilitate the pursuit and attainment of the established goals or objectives. When respiratory home care is pro-

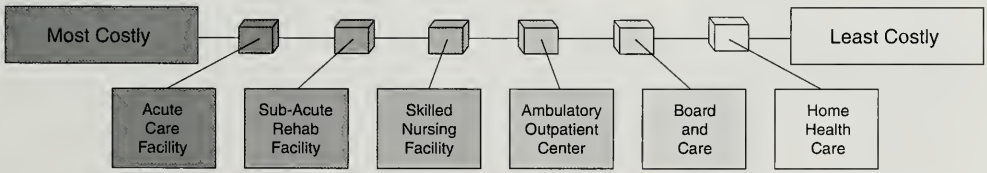

Fig. 1. The concept of the healthcare continuum. Movement along the continuum can be in either direction as need arises.

vided, the chronic nature of the underlying condition often presents the HME/RT provider with the challenge to reduce the frequency of re-hospitalization. Accordingly, the goal or objective would be to improve the patient's ability to manage symptoms and control the triggers that contribute to acute exacerbations. In this respect, the actions or interventions of the home respiratory therapist should promote self-care and self-reliance on the part of the patient or family member. Accordingly, the most valuable and sought after expertise in a home respiratory therapist is the ability to teach the safe and proper use of respiratory equipment in such a manner that long-term adherence to the prescribing physician's prescription is the rule rather than the exception.

Throughout the process of providing home respiratory care, it is essential that the patient's response to the plan be monitored and, as appropriate, that the prescribing physician be notified of the progress toward the achievement of established goals. Often, it may be necessary to revise or modify the plan, especially if assessment feedback indicates that the established goals were unrealistic. Such revisions to the plan of care, if and when indicated, should likewise be discussed with the prescribing physician. Although physician participation in formulating individualized home care treatment plans has become more prevalent in recent years, it is still not as universal as it should be. Recent efforts to incorporate the subject of home care into medical school curricula and postgraduate training programs is certain to heighten further the role of the physician in establishing realistic home care goals.[21] Moreover, the growing trend toward expecting HME/RT providers to obtain accreditation[22] or state licensure[23] is certain to promote further a standardized approach toward providing high quality, cost-effective respiratory home care.

Summary

Healthcare policymakers, governmental and private alike, are now faced with an enormous challenge. Demands for improved access to cost-effective and high quality healthcare are emanating from all segments of our society. Clearly, the traditional model of admitting patients to an acute care hospital as a first-line intervention is losing favor. Although there will always be a role for acute hospital care, utilization of this high-cost setting must be better managed if runaway healthcare costs are to be brought under control. The concept of moving patients along the healthcare continuum as their response to treatment reduces the acuteness of their condition is rapidly gaining support, especially with third-party payors. Accordingly, home healthcare providers, including those offering respiratory home care services, can expect to see an increase in the number of referrals they receive. HME/RT providers must actively promote the benefits they can offer in terms of high quality, cost-effective outcome. It is not unrealistic to suggest that the savings realized by reducing an acute hospital stay by 1 day can easily cover the costs of providing respiratory home care for 4 to 6 weeks. One can only hope that such compelling arguments, occurring at a time when healthcare reform and restructuring is a national priority, will translate to more equitable reimbursement guidelines for respiratory home care providers. The past practice of only reimbursing for equipment and supplies fails to take into account the vital role played by the home respiratory therapist.

Home respiratory equipment and supplies, while an important component of managing chronic respiratory disease, are only effective if used safely, properly, and in compliance with the prescribing physician's intentions. The use of skilled and dedicated home respiratory therapists to train patients, monitor and assess outcomes, and communicate with the prescribing physician ensures optimum results. It is time for reimbursement policies to recognize this vital role played by home respiratory therapists.

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Dunne Discussion

Hill: That was a nice review, Patrick. The perspective I have is that of someone running a respiratory unit in an acute care hospital. Our focus is to try to rehabilitate patients as much as possible and, ideally, to return them home. I've had more frustration than success in getting chronic ventilator-dependent patients home. The main reasons are ones that you spelled out clearly—the need for an appropriate patient and adequate caregivers. Often, these patients are just too sick to allow discharge home or there are neuropsychologic problems that make it unrealistic. Also, their family situations just aren't amenable. I find that you really need, at a minimum, two dedicated daughters who live nearby to make discharge home realistic. Some of you may have noticed the fact that I have two daughters. That's not an accident! Another impediment that we often run into is inadequate coverage by third-party payers. When we don't have adequate family support, the third-party payers do not provide adequate caregiver support. They want skilled caregivers in the home; they won't pay for unskilled people, and yet they will only pay for a small amount of coverage by skilled people. The cost for skilled caregivers for around-the-clock care amounts to about $100,000 a year. Third-party payers cover only a small part, and it becomes impossible to send these patients home.

Dunne: I agree, and I think you've stated the challenge for providers of mechanical ventilation in the home quite well. A support system is indispensable, plus discharge planning and proper support from the hospital as well. The problem that we have is that the reimbursement system is archaic—mired in the past. Reimbursement for a mechanical ventilator is, on average, about $500 a month. A second ventilator may be approved by the payment source for ambulation, possibly mounted on a wheelchair, but that is not always a sure fact. Public funding sources represent the biggest challenge of all. This partly explains why you see only certain companies providing mechanical ventilation in the home, and they tend basically to hope to heaven to break even. Believe me, it certainly endears them to acute care hospitals because they are willing to accept referrals for home mechanical ventilation. So it's really important for HME/RT providers to judge their patient/payer mix to see how many home mechanical ventilation patients they can reasonably handle. The uncertainty of adequate reimbursement, unfortunately, does undermine the other important considerations you mentioned, Nick.

Bach: I think Nick (Hill), when you talk about 24-hour care, you're talking about the more severely physically disabled individual. I want to add to Nick's comments because I have the same problem. I have patients who were coerced into chronic care institutions because of the expense of home nursing care and the failure of third-party payers to cover alternative attendant care. I have self-directed, intelligent patients who are capable not only of learning how to take care of themselves and their ventilators and all the rest, but they also know much more about it than the nurses from the nursing agencies sent to care for them. These nurses follow the regulations of their agencies instead of listening to the patient. I think it's very important that everybody understand that when a self-directed patient without family support directs his home care (with an attendant), tremendous amounts of money can be saved, and the patient gets much better service. The agency nurses are not going to want to drive the patient around or water his flowers; whereas, if the attendant is hired by the patient, then the attendant follows directions from the patient. Invariably patients who have this kind of system say that it's infinitely superior. It's also much, much cheaper. We published a paper in *Chest* last year[1] studying about 30 severely physically disabled people who were 24-hour vent users with no family support since the late 1950s and who would still be institutionalized to this day if they hadn't picketed New York City's Town Hall in 1979 to re-enter the community with client-administered home attendant care services. In 1992, the institution in which they had spent over a decade was getting $718 per diem for ventilator patients. At home, however, the total cost of their care was less than $240 a day. This has been going on now since 1979. So you can imagine the millions of dollars that have been saved for just vent users in this one agency—plus their care is much better. This is sponsored by Medicaid in New York City. It would be appropriate for third-party payers in general to pay for client-directed attendant care when the rehabilitation physician certifies that the patient has been adequately trained and can take responsibility for his own care.

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Dunne: If you recall the continuum slide that showed the acute care, through self-care at home (Fig. 1 in paper), the cost of 1 day of acute care—whatever institution is paying for it—could cover the costs of anywhere from 4-6 weeks of home care. The logic of that has yet to be fully appreciated by many payers—government and private alike.

Celli: I believe that the highlight of your presentation is that continuum, which to me suggests one thing—we're witnessing the demise of independent health agencies. It doesn't make sense to have different, fragmented care agencies all pulling to one single link in that chain, as opposed to an integrated system in which the payers have to respond to this patient, whether he's at this end or that end. My question to you is, How is Kaiser handling that? How are the big HMOs handling that? It seems to me that if I had an HMO that could care for the patient, I would just place him at a specific spot in your link where he would be benefited, both in healthcare and cost.

Dunne: I really appreciate the focus on the financial challenges because that is something that must be addressed before we get into the clinical management and the challenges of the equipment handling. I think what you are suggesting here is basically the concept of a network, in which various providers work together in a community and agree to x number of dollars to care for the patient, per episode, versus each provider billing separately and being paid a fee for what they do. The current system is fragmented and not especially efficient. In terms of what the HMOs are doing, I think it's no surprise that managed care is going to be a dominant part of the healthcare restructuring. It already already is. It's estimated that in 1991, 40 million Americans were members of HMOs, and that number is expected to increase about 15-25% a year. So, more and more Americans are joining managed-care organizations. Kaiser is one of the more definitive managed-care organizations, it not only manages care but they do so using their own hospitals and staff physicians. Newer HMOs lacking the longstanding tradition of Kaiser typically arrange for certain services under subcontract with other independent providers. Many HMOs have agreements with HME/RT providers. As HMOs focus their attention on controlling the utilization of acute hospital care, it is becoming obvious that other sites of care will be needed. Home healthcare will certainly become more attractive.

Make: There are three organizations seeking to assure optimal care in the home. First, JCAHO, a more recent player in this regard, is trying to assure the quality of home care. Second are hospitals and managed-care organizations; and third are the professional organizations. I think JCAHO is usually behind the times. They respond to social and community pressures. I'm concerned that the hospitals are not taking a large enough role in assuring the quality of care. Many hospitals do have their own home care organizations, but they are in the

minority because they don't have the resources or the captive patients to make home care cost-effective. I think professional organizations have not necessarily done a great job, either, because they've mostly been focused on acute care. In the future, what do you see as the roles of those three groups in improving the quality of care in the home?

Dunne: There is no question that all three entities have an interest and role in promoting high quality home care outcomes. With respect to the JCAHO (the Joint Commission on Accreditation of Healthcare Organizations), there is currently under way a major remake of the accreditation process itself. To more fully assist healthcare providers and organizations in the delivery of high quality care, the concept of performance-focused accreditation is being introduced. Under this paradigm, organizations providing a defined healthcare service should continually monitor and evaluate key processes used to deliver the intended service. The effectiveness of a key process is ultimately determined by seeing how closely the actual outcome measures up to the intended or expected outcome. This shift toward linking the voluntary accreditation process with continual self-assessment and quality improvement will address many prior shortcomings with the accreditation process.

The role of the hospital and, for that matter, that of the prescribing physician as well is slightly different in nature. Hospitals and other sources should make referrals for home care services to organizations that consistently demonstrate high quality outcomes. When referral sources recognize HME/RT providers with reputations for high quality services, it encourages other home care providers to make a similar commitment. And, finally, there is no question that professional organizations have a major role and responsibility in assuring quality home care. For example, the AARC is actively working with JCAHO in the areas of home care and hospital accreditation by assisting with the formulation of reasonable and realistic scoring guidelines for performance-focused accreditation standards. In a related endeavor, AARC is also helping define and describe quality respiratory care outcomes through the development and promulgation of clinical practice guidelines,[1-4] consensus statements,[5,6] and standards of practice.[7]

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Sortor-Leger: I think it is important to distinguish between long-term acute and rehabilitation, especially in regard to ventilator dependency—whether it is the COPD or neuromuscular patient. I think the purpose and function of these units is very different. The main goal of a rehabilitation unit is to return a patient to society as independent as possible. All education and training is focused toward this goal. In long-term acute, I think the goal is more toward futher medical support and, as the term implies, longer care and less intense therapy. I would envision some patients moving from long-term acute to rehabilitation.

Dunne: The "long-term acute care" phrase was mine. I personally do not have a problem with the term rehab.

Sorter-Leger: Maybe we need both terms.

Dunne: The difficulty with the term rehab is that certain facilities such as those of a Vencor or Integrated Health Services are classified as DRG-exempt and reimbursed under the existing model along the lines of an acute hospital. When you call yourself a rehab facility, you shift over to another reimbursement model, although the same activities are occurring. So, they're not really calling themselves rehab facilities because of reimbursement issues. But

regardless of what we call a site where subacute care is provided, the goal remains the same—to provide care at a level that is appropriate to the patient's condition and to reimburse accordingly. This is the whole idea embodied in the notion of the healthcare continuum, reimbursement obstacles notwithstanding.

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Current and Future Role of Respiratory Care Practitioners in Home Care

Sam P Giordano MBA RRT

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Introduction

When I began to prepare this paper, I again searched the literature that I have followed for years, and again—even with the growth, the need for 'numbers,' and the threat of increased governmental scrutiny and involvement—I found few data on the home care of respiratory patients. This dearth of information forces a closer analysis of the forces that are driving our healthcare system toward increased utilization of home care and a delineation of the present and potential roles and responsibilities of respiratory care practitioners (RCPs) employed in the home care setting.

Why Change the Settings for Healthcare Delivery?

The reasons for shifting the emphasis from respiratory care services rendered in the hospital to respiratory care services rendered in less expensive care settings, such as the patient's home, are amply documented. The Health Care Financing Administration (HCFA) has projected that healthcare expenditures in the United States will exceed $1 trillion by the end of 1995.[1,2] Further, we know that ventilator-dependent and other technology-dependent patients can be cared for in the home at much less expense.[3]

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The Challenge Is To Balance Cost and Benefit

Managed Care & Site Neutrality

Although it is generally understood that acute care settings represent the most expensive care venues in the current healthcare delivery system, it is not universally accepted that healthcare costs specific to the support of pulmonary patients can be reduced by providing access to respiratory care services in the home. Payers are concerned not only about current and projected costs in the delivery system as it now exists but also about the anticipated costs of adding additional benefits in alternate care settings. The concern is that utilization will increase simply because the benefit of home care has become available. This concern has been expressed time and time again by members of Congress and Health and Human Services officials, over a period of several administrations.

Recently, efforts have been renewed to decrease the number of admissions to acute care facilities and to move patients out of those facilities more quickly. The driving force behind those efforts has been the need to reduce costs and the mechanism has been managed care. Managed care plans are usually paid for on a prospective rather than a fee-for-service basis. This means that healthcare providers now not only are concerned about the patient's health but also have a financial incentive associated with the cost of treatment of patients enrolled in managed care programs, such as health maintenance organizations (HMOs). According to the Group Health Association of America, managed care enrollments have increased to represent more than 41 million subscribers.[4] Because hospitals are an integral part of all managed care organizations, they now have an incentive to shift services away from inpatient delivery in order to constrain costs associated with supporting HMO subscribers. This movement provides RCPs with the opportunity to further expand the integration of hospital-based services with community-based services. This concept of integration, commonly called seamless care, holds a great deal of promise as we, on the provider side, attempt to meet patient needs while constraining costs. Even though policymakers in Washington have been reluctant to add benefits, given their understandable concern about the federal deficit and the potential for overutilization, some third-party payers in the private sector have come to the realization that site neutrality is an important aspect of managed care if costs are to be controlled without compromising the quality or quantity of care.

Utilization

Another facet of cost constraint relates to utilization. Shifting to the least expensive care venue consistent with the patient's condition can curb costs, but we must also be aware that the volume of services offered is a major contributor to cost. In 1992, Medicare's claim volume increased by almost 9%, whereas the number of Medicare beneficiaries grew by only about 2%.[1] That increased claim volume increased Medicare spending by more than a half-billion dollars. Thus, the system must contain utilization controls if we are to bridle healthcare spending. From the standpoint of respiratory care, Clinical Practice Guidelines[5-9] are a step in the right direction. Prior to the development of the Guidelines, many of us had our awareness and sensitivity to utilization and overutilization heightened by the proceedings of the Sugarloaf Conferences.[10,11] Those proceedings described overutilization in what was in respiratory therapy and indicted the profession for not having utilization controls in place. Since that time, the profession has responded with relatively unsophisticated techniques, such as automatic stop-treatment orders. I believe that this experience of almost two decades positions us well for what must now be done.

How Do We Meet the Challenge?

Traditional Thinking

The system must now meet the challenge of balancing cost against benefit. The traditional approach, indeed the more heavy-handed approach, is simply to decrease benefits, and, indeed, such an approach may be forthcoming as a part of health reform. Even so, without adequate utilization controls for the benefits that survive, the system will most probably again increase expenditures by increasing utilization. Another way to decrease costs is to tighten eligibility requirements. Why not just make it tougher for patients to qualify for a given benefit?

In this way, the system can exclude many patients who prior to the rule changes would have qualified for benefits. While making it tougher to qualify will, most assuredly, curb healthcare spending, we may be forgetting the reason for the healthcare system in the first place—the need to care for the sick. In my opinion, these two elements of traditional thinking do not bring about a balance of cost with benefit.

The New Thinking

Perhaps, then, we can, through managed care and utilization control, offer services with the appropriate balance of cost and benefit—by shifting patients to the least expensive care venue consistent with their condition and employing controls, such as Clinical Practice Guidelines.

Who Will Provide the Care?

The Impact of the Respiratory Care Practitioner

There are those who believe that other caregivers already recognized for reimbursement in the home care setting can deliver the necessary care. I believe this to be more wishful thinking (ie, if you buy into this belief, additional benefits will not be needed). However, a key player is missing from the home-care scenario—the qualified RCP who can coordinate all of the resources necessary to adequately maintain a particular patient in the appropriate care venue. Studies[12-14] have shown that RCPs who are allowed access to pulmonary patients in the home have a positive impact. The RCP accomplishes this by coordinating the resources necessary to properly support the patient and by assessing the patient, the patient's environment, and the family caregivers and by feeding this information back to the case manager and/or the attending physician.

The fact is that RCPs are needed to perform the elements of their scope of practice in the home[15] just as much as they are needed in the hospital. This is not a criticism of other health professionals because practitioners of other disciplines have not received the training necessary to successfully execute all of the elements comprising our scope of practice.[16] Both the South Hills[12] and Connecticut[13] studies recorded a significant decrease in length of stay and frequency of hospital admission after home care patients with pulmonary disease were allowed access to an RCP. Prior to implementation of RCP services, nurses and other healthcare providers supported these patients in their homes. From those studies, one can infer that the RCP made a positive impact by monitoring these patients in their homes. This monitoring allowed earlier identification of acute exacerbations and, thus, earlier interventions. A patient's condition was not allowed to deteriorate to the point that hospitalization became necessary but rather less expensive interventions stabilized the patient short of an expensive hospital stay.

Only RCPs are formally trained and qualified via competency testing to perform respiratory care procedures.[17,18] This does not mean that others cannot be trained and subsequently demonstrate competency. It merely means that no others are choosing to do so at this time. I speculate that the reason for this is a form of 'natural selection.' All of us as RCPs aspired to be RCPs. We didn't aspire to be nurses; otherwise we would have gone to nursing school. We didn't aspire to be physical therapists; otherwise we would have sought training in that special area. By the same token, these highly qualified professionals do not aspire to be RCPs; so, it seems illogical to assume that just because nurses and physical therapists are recognized for reimbursement in home care that they have the expertise to render respiratory care in the home. Such illogical assumptions tend to occur when policymakers cross over the line into patient care. The illogical assumption may make the arithmetic look good, but , in the long run, (1) the patient loses and (2) the system may lose because hospital admission rates may be higher for respiratory patients who do not have access to the services of a qualified RCP. Respiratory care is not a hobby; it is a profession and cannot be mastered without devoting full attention to it.

If other healthcare providers are not positioned to render respiratory care services in the home, then it follows that RCPs must be allowed access to these patients. Let me repeat—RCPs are the only healthcare providers trained and qualified by testing to perform respiratory care, and the few studies available demonstrate that when an RCP is allowed to intervene in the home a significant savings can be realized. The savings are more than enough to com-

pensate for the expense involved in rendering the service because those savings are derived both from decreased length of stay and decreased admissions. I believe that with proper utilization, this particular benefit can more than pay for itself. Because of concerns regarding utilization (or overutilization), there is still reluctance on the part of third-party payers to embrace this benefit on a universal scale. We must now redouble our efforts to demonstrate the cost-effectiveness of employing RCPs in the home environment. With the continued move toward managed care and capitated payment, this actually becomes an easier 'sell' than it would be were the old cost-plus reimbursement system still in place. Because managed care provides financial incentives, in effect, to minimize the use of acute care services, it follows that greater access to services in the home is required if we are to decrease our dependence on acute care.

An Expanded Role for the Respiratory Care Practitioner

We must expand the role of the RCP to go beyond that of a 'high-tech' caregiver and task performer. Traditionally, we have also occupied the role of care coordinator when allowed to do so. Indeed, many of the activities we undertook in response to the Sugarloaf reports were aimed at a better coordination of resources, to bring about an appropriate match between respiratory care resources and need, regardless of the direction that healthcare reform takes in this country. The new system must require health professionals to take responsibility for controlling utilization as well as for rendering care. Going one step further, when working in the home, RCPs must position themselves as care coordinators for those patients who are primarily respiratory patients. This means that RCPs must coordinate all resources, which include equipment and other caregivers, in such a way as to bring about a proper balance of cost and benefit.

Additional Skills and Responsibilities

Assessment of the patient's physical condition must be expanded to include assessment of activities of daily living (ADL) and relevant psychologic and sociologic factors. Heretofore, with most of our efforts concentrated in the acute care setting, those aspects of assessment were not as important as they are today. We must also be able to assess family caregivers because these individuals can play an important role in keeping chronic patients well enough to avoid hospital admission. We must therefore be educators, not limiting our own knowledge to the patient's disease but expanding it to include the patient's physical environment, disease prevention, nutrition, and other aspects that contribute to the patient's overall well-being.

The perfection of critical thinking skills[19] spawned in the acute care environment looms at the top of the list of needed changes because the new healthcare delivery system must demand from all providers that they constantly search for better alternatives for patient support that promote more desirable patient outcomes and/or less expensive clinical interventions.

There is no doubt that RCPs are underutilized in some care settings. It is now time to explore and introduce additional duties into the scope of practice for qualified RCPs, such as the administration of medications by routes other than aerosols. To me it makes no sense that a licensed vocational nurse with 1 year of training and a postgraduate drug course can be qualified in many states to administer intramuscular and parenteral medications, but a credentialed RCP with at least an associate degree cannot take the same drug course as a postgraduate and become qualified as well. This is one example of the many inconsistencies in the current healthcare delivery system.

Respiratory care practitioners working in the home environment will perform physician-ordered assessments and use Clinical Practice Guidelines to assist in monitoring utilization of our services. Treatment protocols are just beginning to come into vogue. Once fully insinuated into the healthcare system, the quality and timeliness of clinical decision making will be aided as defined in the treatment protocols. Respiratory care practitioners will then assess, treat, and educate patients on behalf of the attending physician. Practitioners will also assist the physician in care venue selection—a selection based on the patient's ability to perform ADLs, the family support network, and access to services needed by the patient. If the patient's primary problems are respiratory/pulmonary, then the care plan

could be coordinated by the RCP. Once the treatment plan and the care setting are identified and all resources coordinated, then the RCP will follow the patient by both telephone and personal visits. Information obtained during follow-up will be shared with the attending physician on a regular basis in order to head off problems before they become serious enough to require hospitalization. The patient's care plan will be reviewed with the physician and revised as needed, based on the practitioner's observations and assessment.

Shared Turf

We must be prepared to share our expertise with other healthcare providers. Many critics of the current U.S. healthcare system cite overspecialization as one of the reasons for high costs. Although specialization is necessary, all health providers, including RCPs, must review their scopes of practice and identify those tasks that involve low risk and minimal complexity, and are easy to learn. Those tasks can then be taught to other providers. When scopes of practice are expanded, greater efficiency and more complete utilization of provider personnel results. All caregivers must, therefore, intentionally blur territorial lines—not to displace other health provider colleagues but rather to complement their efforts.

Overcoming the Factors that Limit Home Respiratory Care

The most important limiting factor that we currently face in home respiratory care is that in the U.S., care follows the dollar. To put it? another way, if there is no reimbursement, there will be no service. We must continue our efforts to demonstrate that home care rendered by RCPs is cost-effective. This is a hard sell because of reasons previously mentioned. However, it not impossible, and, with the movement toward managed care, this effort is 'picking up steam.' Demonstration projects must be developed in even greater numbers than in the past—projects designed to demonstrate the economic and clinical impact that RCPs can make on home care patients. The few studies[12,13] that have been done have not failed to demonstrate a cost-savings stemming in part from decreases in hospital admissions. Our challenge now is to collect larger quantities of data in order to convince even the most skeptical policymaker of the benefits of home care.

Besides reimbursement and demonstration projects, we need physicians and consumers to help decision makers understand that current coverage provisions are inadequate from the standpoint of the respiratory care patient. Indeed, current reimbursement is discriminatory when viewed from the patient's perspective—if you have a chronic respiratory disease, you do not have access to a formally trained and credentialed respiratory care provider in the home but must be admitted to a hospital to receive their care, pay out of your own pocket, or have an insurance policy that allows for this coverage. On the other hand, if you fall and break your hip, once you have been discharged from the hospital, you are assured of the services of a qualified physical therapist. That's just one example of the discrimination faced by respiratory patients every day. Consumers and physicians are the most credible when it comes to describing the need to have access to RCPs in the home because neither would be perceived as being self-serving in advancing such an argument.

Another limiting factor that is currently being addressed but will take time to overcome is the fact that RCPs need to broaden and deepen their education to prepare themselves to assume greater clinical and nonclinical responsibilities on behalf of the attending physician and patient (a need not unique to RCPs). It is always easy to say that we want more—without doing more—but the fact is that the only way that we can be deemed qualified to assume additional responsibilities is to acquire the education necessary to inspire confidence in decision makers. Our curricula and program structures are changing to position the profession to be fully franchised in the healthcare delivery system of the future. Practitioners in the current work force must step up and make individual efforts to acquire the education necessary to perform in the expanded scope of practice. Many of our colleagues have done and are continuing to do this.

A Model for the Future

Consider the model, shown in Figure 1, as an example of what could be done by integrating acute

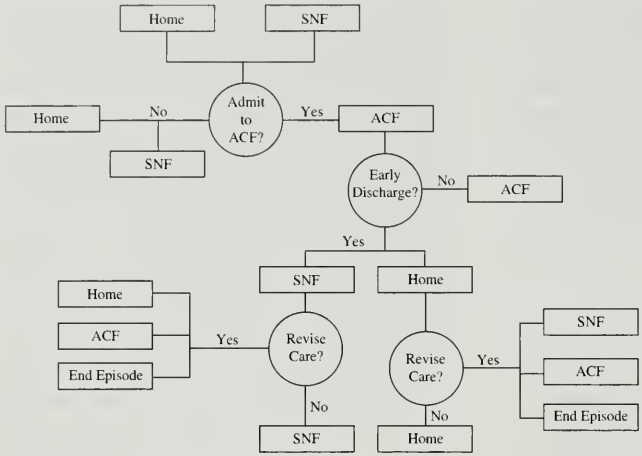

Fig. 1. Decision model illustrating the concept of episodic care. The model assumes that early discharge connotes the need for continued care until the episode ends. The model (1) uses utilization controls for acute care in all three care settings; (2) recognizes the "care episode"; (3) decreases administrative costs by using already existing utilization controls; and (4) decreases costs by 'downshifting' patients as soon as possible and eliminating Medicare 90-day requirement because of qualified caregivers in skilled nursing facilities and home. The circles represent decision points determined by the physician's re-evaluation of the patient. SNF = skilled nursing facility; ACF = acute care facility. See text for more detail.

care and community-based services—a system of seamless care. All care settings are recognized as part of the system overall. Respiratory care practitioners could then think in terms of episodic care, that is to say, an RCP would have responsibility (delegated by the attending physician) to follow a particular patient throughout his or her episode of care. This would mean that the practitioner would follow the patient regardless of the care setting, always assessing, always coordinating resources, treating or arranging treatment, and monitoring the care plan. This RCP would work for a managed care organization that would have acute, subacute, and long-term components. The goal of the episodic care manager would be to make sure that the patient is shifted to a care venue commensurate with the patient's resource needs. If the patient is admitted to the hospital, the goal is to provide the care required and then discharge the patient as soon as possible and follow that patient to the next care venue, which might be a subacute care unit, a skilled nursing facility, group facility, or the patient's home. Once in the next care setting, the practitioner-coordinator would continue to work at the behest of the attending physician to assure that care continues appropriately during the episode. The frequency of order changes and assessments would be decided by the attending physician, but there would be no 'slippage' because of the hand-off from the acute care setting to another setting. Indeed, all services would be tied together by that one RCP responsible for the particular episode. Of course, once the patient progresses to the point where the episode has ended, the attending physician 'discharges' the patient, and the RCP-coordinator is assigned to follow another patient. This is only one model for RCP utilization. Speculation suggests that hospital utilization by respiratory patients could be decreased in terms of length of stay and number of admissions if patients were not required to be admitted to a hospital to have access to respiratory care services and, once in the hospital, be forced to stay until able to function almost independently because of the lack of home services under the current system.

In Closing

There is no doubt that many respiratory care practitioners are proving their worth today in the home care environment. We will, as a profession, increase our value to the healthcare system by expanding our scope of practice in a way that allows us to render more comprehensive support to patients outside of the hospital. Ultimately, third-party payers will recognize the value of reimbursement for the professional services component of respiratory care because it makes good economic sense to do so. The challenge to the profession is to be prepared to fulfill this important role when the opportunities manifest themselves.

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Giordano Discussion

Bach: Sam (Giordano), I just have to say that I couldn't agree with you more about the respiratory care practitioner, the respiratory therapist—as a care coordinator. We all need to document our experiences to show that RCP-provided care can be better and less expensive. Many times I've been able to manage, without hospitalization, patients with acute respiratory failure, especially patients with neuromuscular conditions who use nocturnal nasal ventilation and who present with respiratory infections and severe mucus plugging. (Anyplace else they would have been admitted and intubated.) I do the safest thing. I send them home with a respiratory therapist, an oximeter, and an exsufflator. The respiratory therapist teaches them that if they desaturate, they may have a mucus plug and should use the exsufflator to help eliminate it. The oximeter also guides them in using noninvasive ventilation for sufficient periods, often 24 hours a day, until the infection is gone. I would not be able to do that if it weren't for my specifically skilled and experienced respiratory therapist. He really coordinates the care, and by keeping patients out of the ICUs, he saves a fortune! I have patients who have had several

colds and presented with acute respiratory failure for several years and would have been hospitalized and intubated each time. So, it is important to get the message out. Our respiratory therapist saves my neuromuscular, spinal cord, and polio patients from ever needing to undergo a tracheotomy, at least as long as they can swallow.

Pierson: It may be early in the conference, but I will bring it up now. We have an inherent problem that's going to have to be dealt with in the scenario you talked about, Sam. You finished by talking about the respiratory care practitioner as a coordinator of care, whose primary value is what is between his or her ears. We have in the United States thousands of people working as respiratory care practitioners who don't meet that description by a country mile. One of the speakers at this conference told me last night that he has therapists in his department who are OJT from housekeeping. Now, how do you reconcile the need for what you just described with the state of things as they really are in some respiratory care departments, and outside of hospitals, and what is today's respiratory care practitioner going to have to do to change into what you described?

Giordano: First of all, I agree with your assessment. We are, fortunately, talking about the future, but it's the near future. Just as your profession will change and retool for this thing, so must ours; and the challenge is before our profession. Either we rise to this level of performance or we cease to exist. The one thing the system doesn't need is highly trained professionals who can only render tasks. You can always teach people to perform tasks and then supervise them closely. We're stepping up. We're reorganizing our educational system. We're raising the entry level, expanding critical thinking skills, retooling, reinventing.

Dunne: Sam, I don't think the changes you envision will be limited to therapists practicing in the home either. The acute care hospital setting will also benefit from this expanded role of the RCP as a case coordinator, as a case manager. I think that as we look at the continuum, probably the biggest challenge will be to change the thinking of the hospital-based RCP—to be a bit more futuristic and to take the blinders off and see beyond just the fact that they're rendering tasks. As respiratory therapists become more involved in subacute, long-term care, rehab, and home care, I think we will begin to witness many examples of the true cost-effectiveness of quality respiratory care provided by qualified respiratory therapists in all care settings. The preliminary data are already quite compel- ling in that regard. Indeed, the whole thrust of the RCP 2001 project and one of the recent educational consensus conferences[1,2] is to broaden the base of skills for therapists so that they are qualified to practice in all of the various sites within the healthcare continuum.

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Kacmarek: I have to agree with both Sam (Giordano) and David (Pierson). The concern that I see with the profession is that we are lagging way behind in what needs to be done to bring our practitioners up to the level where it is even feasible to conceive that they'll be able to fill this role. We have not made the decision to change our entry level. Sam indicates that we will, but when? There is no change in the fact that half of the practitioners entering the profession of respiratory care enter as certified techs who are not going to be able to find positions. In Boston, very few of the major institutions will even hire graduates from those programs because they are not skilled to perform the tasks expected. The decision that we as a profession must make is that we want this described player. You and I and Sam and a few other people's saying it, is not going to make it so. Unless the rest of those involved in respiratory care decide some drastic changes must be made, we will never come near developing practitioners with the profile that Sam has provided. Today, if we look at other individuals involved in the healthcare system, who are making decisions, frankly, I don't think we have a chance of assuming this level of position, based on the average common denominator that exists in the respiratory care profession, unless change is imminent.

Giordano: Can I respond to you? While what you say may be true, I think that what you have to understand is the motivation for change. The motivator is coming right down at us. The fact is acute care facilities are downsizing and will continue to downsize. There's much too much capacity in the acute care venues. If our people, our RCPs, are trained and exist in an organization in which a lot of people above them do their thinking for them, they will fall by the wayside. There's nothing like having a genuine threat on your future job security to shake the tree, to the extent that you're always going

to have people who are not motivated to do more. The fact is it's these individuals who get out. These individuals are getting out. So, we'll have some thinning of the ranks, just by the fact that they were never 'dug in' that deep in the first place, and they want to find some easy thing to do. When they look around for job opportunities, they'll find that only people possessing the qualifications described are going to be in demand. That will motivate us. We're not going to get into a lot of this inside the profession—you know, we think it's this, and we think it's that—because, really, the forces at work are outside the profession, and they are going to motivate the right people. Will we have more or fewer technicians? I say we'll have fewer technicians, but whether we have more or fewer practitioners in terms of the higher level people, I think there will be more of those individuals. But, certainly we have a long way to go. If you take that snapshot today, you're going to say, OK, we can't turn these masses of unqualified people loose. Well, I doubt you're going to need large numbers, comparatively speaking. We don't need the new breed of practitioner in place tomorrow, but, certainly, we need to get busy. And we need to move quickly.

Leger: I think it's really important to define clearly the job of practitioners in the home. You quoted in one study the need for a visit every week from a healthcare professional, one week the nurse, the next week the respiratory therapist. What kind of job did they do in the home? If they were providing treatment to a patient as if they were in subacute care, I understand. If it was just to supervise, I think it is excessive. We probably have very similar patients in our home care institution in France, and we can see the patients when it is necessary (if the patient, physician, or caregivers call with specific problems) and systematically once every 3 months. I think it is important to be responsible in this kind of an organization, but visits can be very expensive, and it is necessary to prove that we have better results with more frequent visits. We need to know, for example, that the treatment compliance is better, the survival rate is better, that the number of hospitalizations decreases. I'm not convinced that visiting a patient every week changes a lot.

Giordano: The study I referred to done by Haggerty et al,[1] and published in *Chest*, had that kind of a schedule. I am not proposing that every chronic pulmonary disease patient needs to have a weekly visit from a respiratory care practitioner. That, indeed, should be worked out within the care plan, and the attending physician needs to adjust not only who will visit but how often according to the patient's condition.

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Leger: Who decided the frequency of the visit?

Giordano: Well, in the paper I'm referring to, this was a set protocol. All 17 patients involved in the study were chronic lung patients, a protocol was followed, and they were visited one time per week by an RN and once every 2 weeks by an RCP.

Hill: Obviously, your talk has generated a lot of comment. I think we're all concerned about what the managed-care future is going to hold for us, how many bodies are going to be paid for, what kind of turf battles are going to be fought, what niche will be left for us. You're describing an expanded role for the respiratory care practitioner. I'm not sure that it's going to end up quite as expanded as you have outlined. I think the role of the respiratory care practitioner in the home will continue to be tied, to a certain extent, to equipment. When I think of situations where a respiratory care practitioner is most needed in the home, it's with the patient who is using a ventilator or some other respiratory equipment in the home, and the visiting nurse doesn't have the training to manage that patient. I rely heavily on the practitioner in those circumstances, not only to maintain equipment but also to keep me apprised on cognitive issues, too. But the COPD patients with multiple medical problems really need a visiting nurse because they have a lot of other problems. They have medication issues that are of major concern. Likewise, in patients with intermittent asthma, you don't really need respiratory care practitioner involvement. I still say that the role of the respiratory care practitioner in the home will continue to be tied to equipment, and I think that role should be expanded. Third-party payers have to recognize that there's a cognitive component to caring for patients who need respiratory equipment, and that component should be reimbursed. Respiratory care practitioners are really essential for the home management of respiratory equipment needs, and their role should include not only maintenance of equipment but also periodic patient assessment and education.

Giordano: Well, I agree with you in terms of the equipment. I didn't mean to imply that we are going to leave that behind—we are going to add to that responsibility, responsibilities that are not equipment-related. Now, as far as utilization of respiratory care practitioners—before-and-after studies[1,2] have shown that

where patients had been seen only by the visiting nurse the introduction of respiratory care practitioners brought a decrease in the hospital admissions.

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Celli: My comment contrasts with Nick's (Hill). I think we're taking a gloomy view. I think respiratory care practitioners could expand tremendously along new frontiers. In rehabilitation, RCPs could take over the centralized pulmonary rehab world from Lionel's B&B Feed, which views the patient as a fragmented bunch of arms and legs. The multifaceted therapists could very well become sleep technicians as sleep technology becomes more simplified. Pulmonary function testing and exercise testing—when they're not doing ventilators, they're doing PFTs. Bronchoscopy—with all the new visual endoscopy technology—I think the respiratory therapist should be part of that. So, I see the intelligent respiratory therapist as a multidisciplined individual who can help with the technology and the concepts that are available to the pulmonologist. I think the task you guys have is to train people to tackle each of these components.

Stoller: Although I agree with you Bart, I think the problem that we face at the policy level is that we're now in the era in which policymakers are looking abroad for models of healthcare delivery to guide current policy in the United States. And when they look abroad, they see many sophisticated countries other than the United States in which patients are cared for reasonably well in their view, less expensively in their view, and without anybody who is called a respiratory care practitioner. Although I think we may 'be preaching to the converted' in this meeting, my question to you is, How do you deal with the policymakers who have that perception of the outside world and are trying to forge American policy modeled on outside plans, where there are no respiratory care practitioners?

Giordano: Well, I think that if you hark back to the origins of this profession, you can see that it really was (as near as I can trace it) the result of a survey that was done in New York City hospitals in 1950.[1] A committee formed by the New York Academy of Medicine with physicians and nurses as members surveyed all the hospitals and identified many problems associated with inhalation therapy, as it was called in those days—a lot of nosocomial infections and those kinds of things. One of the recommendations was that they organize a separate group of people to become expert in this area to solve some problems. Well, if you don't look for the problems—they aren't found until they are so big they can't be ignored. Do they have the same problems that we had 43 years ago, that don't really exist on a large scale? You also want to look at their hospital utilization. In Japan—we were just talking about large state rehab hospitals—length of stay in an acute-long stay hospital is 25 days. I think the length of stay in Japan is about 38 days for an acute care hospital. For a similar hospital in the U.S., length of stay is about 7 days. There are other ways of taking care of the patient, but I guess the question is, Is that the best way? Is that the optimum way? Their concern is now about hospital utilization in many of these countries not because of the cost—although concerns about costs are beginning to creep in, especially in the Canadian system—but, they're concerned because they just don't have enough capacity. In the People's Republic of China, their length of stay, I think, goes beyond 30 days. And their concern is not that it costs too much, but that they just don't have enough beds for all these people. So, I think what we're seeing is more utilization in what we consider to be high cost areas but because of the differences in their system, they don't recognize it that way. The total cost of healthcare in Japan is, I believe, 185 billion dollars this year. We would love to have such a low rate here.

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Hardy: This has been a stimulating discussion. We're all talking on both sides of the fence. You started your lecture by saying you were predicting the future; and there was no right or wrong. Because it's the future, we don't know what the right or wrong will be. We've heard the doom-and-gloom side and the optimistic side. One of your slides was "Cost Is What Determines Care," and I think all of us see that the dollar is really king here. Everything that's going on in healthcare right now and the move to restructure all of our acute care facilities is highly driven by the dollar, and is going extremely fast. In hospitals across the country, consultants like Booz-Allen come in to make their major assessment of a hospital. One of the first organizations they always attack is respiratory care, and downsizing of everybody's respira-

tory departments is happening quickly. On the one hand I agree with Bart (Celli). I can see all the opportunities. I'd add airway clearance to your list of things in which an RCP could play a major role. However, we don't have adequate data right now to support those ideas and visions. And we can't acquire it fast enough. We can't hope to get this information into reputable journals and published fast enough to keep up with the restructuring and the cost-containment that's going on right now. In addition a large group of RCPs don't match the same level of expertise or vision as those who are sitting in this room and who are usually at the top of respiratory care organizations.

Giordano: If I may, I tend to stay with the more optimistic side. I can't say that we're not going to hit some rocks in the road, but, Number 1, you refer to a consulting company that has no hard data. What we have is uninformed decision makers, which is part of the reason why the system is the way it is. The other thing is that our people are used as utilization controllers. Even though we don't have a lot of data, as you say, and you're correct in pointing that out, we do know that certainly one of the major cost drivers is utilization. Positioning respiratory care practitioners as utilization controllers, as agents of the physicians, has been and continues to be a very good thing. The therapist-driven protocols are coming out now, and we're seeing the decreases in utilization without the decrease in the quality of care. Indeed, we're seeing better clinical outcomes because the quality and timing of the decision making is that much better. So, yes, there are some negatives, but we need to balance those with some positives. Certainly we can't forget that our people, the respiratory care practitioners—like the physicians, the nurses, the OTs, and the PTs—are going to have to step up and get in with this new 90s kind of thing in health reform, which talks about money just as much, if not more, than it talks about the care for the patient.

Make: This discussion is great! How can we cut down the amount of time for the presenters and leave more time for discussion? Sam raised many very important points that I'd like to follow up by suggesting that this conference take a slightly different direction. These conferences usually produce comprehensive journal issues with articles and discussions but often do not make specific recommendations. What we've talked about is the importance of obtaining data to assure a role for specialists in respiratory care, and that the data need to be outcome-based. There are many types of outcomes, including quality of life, clinical outcomes, and the economic issues that we've discussed. We raised a question about whether nurses could provide services traditionally performed by respiratory therapists in the home. Do respiratory therapists define themselves too narrowly in the home? Nurses define themselves more broadly in the home. We should rephrase the question as "Can RCPs provide services traditionally performed by nurses?" The AARC is taking the lead in trying to redefine the role of the respiratory care practitioner in the future. One of the recommendations that might come from this conference is that we need more information about the role of the respiratory care practitioner in the home. Obviously, we think that respiratory care is best provided by a specialist (RCP) in the home, rather than a generalist (the nurse). But, there is no information to suggest that this is indeed the case. I suggest that we recommend that the AARC fund studies to evaluate the outcomes of models where RCPs provide respiratory care in the home setting.

Sortor-Leger: I would like to make a comment regarding the need for nonprofessional care in the home for ventilator users. We have many technology-dependent patients in the U.S. whose financial resources are very limited or nonexistent. Many of these people remain hospitalized or in intermediate care centers that are expensive and do not serve the purpose of reintegrating these people back into society. In addition, many of these people are able to direct their own care, given the opportunity and training, allowing them to live outside a healthcare institution. A possible role for the respiratory therapist in this scenario is to supervise the nonprofessionals and/or provide the training. I think this is a more economical solution, which would allow us as a society to provide more hours of care to a greater number of ventilator users.

Make: We've talked a lot about specialized care in the home for ventilator-dependent, high technology patients. Let me make sure that we don't get too focused on this specialized population. We need to remember that the vast majority of patients in the home who get lots of visits by respiratory care practitioners are receiving less complex, more traditional therapies, such as simple oxygen therapy. What are the outcomes of care provided by RCPs in these patients?

Hill: Yes, but most simple oxygen therapy is provided by companies who deliver oxygen but don't provide respiratory care services (ie, education).

Hoffman: I want to endorse what Barry Make said about outcome studies. It is important to remember that respiratory care focuses on a single system. That's not problematic in a hospital because many patients can be seen in one workday. When care is provided in the home, other factors

need to be considered. It's time-consuming to travel from home to home, and patients have multiple needs. Practitioners with a single-system focus may be viewed as less cost-effective. Maybe the respiratory care curriculum should expand—become more broadly focused. Another approach would be to critically evaluate those things respiratory care practitioners do the best that would merit their making a home visit.

Giordano: You're right on target. With all healthcare providers, there's going to be and should be some overlap—where cross-training can be economical and skills can be kept up. Certainly a respiratory care practitioner sent to intervene with a patient should be cost-effective just because of the things that have to be looked at and done for that particular patient. Not every patient who receives respiratory therapy needs therapist intervention, but when the problems or issues involved with that patient are predominantly respiratory care issues, then it might work better. Certainly there are going to be those patients for whom respiratory care is a secondary thing; also there must be some 'cross-fertilization' in terms of responsibilities shared among other healthcare providers, nurses, PTs, RCPs. This improves cost benefit for all caregivers including RCPs.

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VIII. The Need for a Study of Supplemental Oxygen for NOD Patients

Introduction

The Nocturnal Oxygen Therapy Trial (NOTT)[1] and Medical Research Council (MRC) studies established and justified the role of home supplemental oxygen for chronic obstructive pulmonary disease (COPD) patients with a daytime $P_{aO_2} < 55$ torr.[1,2] These two studies showed better survival in patients receiving oxygen for 18.6 versus 12 hours per day (NOTT study) and greater than 15 hours per day versus untreated patients (MRC study). Thus, survival in oxygen-treated hypoxemic COPD patients is better than in those receiving no therapy, and use for more hours/day is better than only overnight or 12-hour use. Home oxygen may also lower daytime resting pulmonary artery pressure (P_{pa}) in such patients ($P_{aO_2} < 55$ torr).[3-6] No large prospective studies have been undertaken to further define the role of home oxygen therapy in COPD since these studies were carried out in the 1970s.

We have just begun to 'scratch the surface' in our quest for knowledge about chronic supplemental oxygen. For example, these two studies, as well as most others, have been directed toward the group of patients with levels of daytime hypoxemia that cause pulmonary artery vasoconstriction and pulmonary hypertension. Can oxygen use affect morbidity or mortality if applied at an earlier stage of disease? Can it help patients with transient hypoxemia under special circumstances such as during sleep or exercise? The following discussion addresses data available on the potential value or efficacy of oxygen during unstable hypoxemia such as transient desaturation occurring during sleep or exercise.

Transient Desaturation

The concept of transient oxyhemoglobin desaturation is a recent one, being made possible by innovations of continuous oxygen measurement, such as ear and pulse oximetry, which measure oxygen saturation (S_{pO_2}) continuously, and the transcutaneous

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oxygen electrode, which measures oxygen tension (P_{tcO_2}). Transient desaturation in COPD patients has been identified during cough, eating, micturition and defecation, and even speaking. Probably of more importance is desaturation during sleep and exercise.

Early sleep researchers identified transient nocturnal oxyhemoglobin desaturation (NOD) during sleep by either serial samples of arterial blood[7,8] or by using forerunners of modern pulse oximeters.[9,10] They identified desaturations lasting to 30 minutes or longer associated with what appeared to be normal breathing. Wynne et al[11] provided a detailed description of desaturation in COPD patients during sleep, which he divided into two types: short episodic (a few seconds to several minutes) and longer NOD (several minutes to an hour or more). The short episodes were frequently related to observed alterations in breathing pattern (eg, hypopneas, apneas, and coughing), but the longer episodes were not identifiable by alterations of respiratory pattern (Fig. 1). Because the latter were of longer duration and associated with elevations in P_{pa}, they have since become synonymous with NOD in COPD. Exercise desaturation was described (relying on similar technology) about the same time.[12] The desaturation appears shortly after onset of exercise and usually worsens as exercise progresses, with arterial saturation returning slowly to baseline upon exercise cessation. The desaturation probably contributes to exercise limitation by chemoreceptor-stimulated perception of dyspnea, but, by no means, is exercise desaturation the most common cause of exercise dyspnea or limitation.

Mechanisms of Transient Desaturation

Numerous studies have demonstrated that NOD is most frequently seen and certainly much more profound during rapid-eye-movement (REM) sleep.[8,11,13] This is the stage of sleep, defined by electroencephalograph (EEG), when most dreams take place. REM sleep is associated with changes in many physiologic control mechanisms, including a shift in parasympathetic-sympathetic balance that results in changes in heart rate, blood pressure, and—more germane to this discussion—respiration. The EEG pattern during REM sleep is similar to the awake one. Skeletal muscle tone undergoes a type of descending block or inhibition of activity due to neuronal hyperpolarization generated in the brainstem. The teleologic reason for this blockade is probably to keep us from acting out our dreams and thus waking or injuring ourselves. Because the intercostal muscles and the accessory muscles of respiration are skeletal muscles, they too undergo blockade and thus lose tone. This results both in a decrease in tidal volume and functional residual ca-

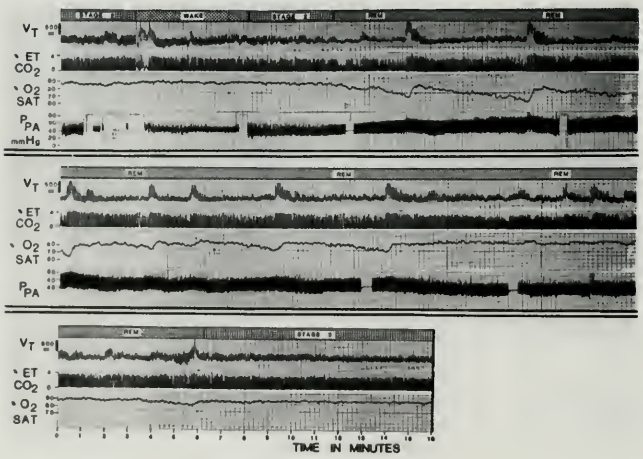

Fig. 1. Continuous tracing of sleep stage, tidal volume (V_T), end tidal CO_2 (% $ETCO_2$), arterial oxyhemoglobin saturation (% O_2 SAT), and pulmonary artery pressure (P_{PA}) during sleep in a patient with nocturnal oxyhemoglobin desaturation (NOD). Shortly after the onset of rapid eye movement (REM) sleep, arterial oxyhemoglobin saturation drops progressively to about 65% and remains low until near the end of the REM period. Alternating hypopneas and hyperpneas correlate with the fall and minimal recovery throughout the REM NOD.

pacity (FRC) and maldistribution of ventilation.[13] Such falls in FRC during REM sleep have been well documented in normal humans and in various disease states.[14-20]

The fall in FRC in normal subjects probably does not affect gas exchange as long as thoracic gas volume remains above closing volume. But, in patients with COPD whose thoracic gas volume resides just above closing volume and at higher than normal lung volumes, loss of intercostal muscle tone would predictably have a greater effect than in normal subjects (Fig. 2). In such patients, the diaphragm is already functioning at a point near maximal contraction, and it may be unable to increase excursion to make up for the accompanying loss in thoracic gas volume.[20] The combination of loss of intercostal muscle tone and the inability of the diaphragm to increase thoracic gas volume may then allow FRC to fall below closing volume. This adds to the number of areas of low ventilation-perfusion ($\dot{V}/\dot{Q}$) already present in diseased lungs by further increasing maldistribution of ventilation or creating microatelectatic areas.[13,20,21]

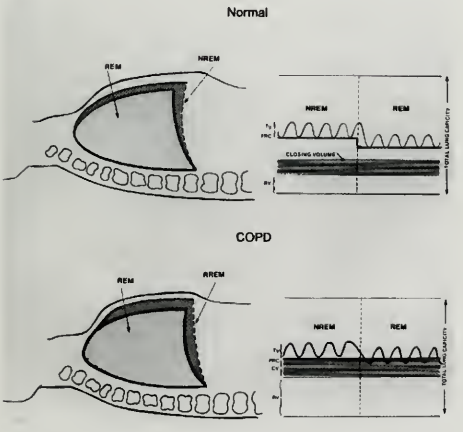

Fig. 2. Schematic representation of one mechanism for nocturnal oxygen desaturation (NOD) in COPD patients. When normal subjects enter REM sleep, functional residual capacity (FRC) falls, but most lung units do not approach closing volume. In patients with COPD, closing volume is just below FRC while they are awake and in non-REM sleep; therefore, the fall in FRC accompanying the onset of REM sleep results in some lung units' falling below closing volume. This results in worsening of $\dot{V}/\dot{Q}$ matching and adds to the hypoxemia of hypoventilation. (From Reference 13, with permission.)

Several additional factors such as the position of the patient's P_{aO_2} on the shoulder of the oxyhemoglobin dissociation curve, the presence of alveolar hypercarbia (carbon dioxide retention), depressed cough and decreased mucociliary clearance, and depressed chemoreceptor ventilatory responses during REM sleep may contribute to the REM hypoxemia.[21] It should be emphasized that these mechanisms are separate from those in obstructive sleep apnea in which the upper airway closes and air cannot enter the lungs. Although patients with COPD can have sleep apnea, it is incidental to the REM-sleep NOD and arises by separate mechanisms.[22]

Are the mechanisms of exercise desaturation similar to those in sleep desaturation? Probably not. The setting for desaturation under each circumstance is different. Whereas REM-sleep NOD is associated with hypopnea,[13,22] a decrease in FRC, and substantial worsening of $\dot{V}/\dot{Q}$ mismatch due to ventilation maldistribution, exercise is associated with hyperpnea and an increase in FRC,[23] with probable mild worsening of $\dot{V}/\dot{Q}$ matching.[24] Van Meerhaeghe and Sergysels examined gas exchange variables in a group of 24 COPD patients and 15 normal subjects.[25] They found that CO_2 retention during exercise in COPD patients resulted from the mechanical inability to increase minute ventilation to the extent that normal subjects did and probably not from differences in respiratory drive. Using the 6-inert-gases technique and indwelling arterial and venous catheters, Dantzker and D'Alonzo simultaneously examined $\dot{V}/\dot{Q}$ matching and gas exchange (shunt fraction, arterial P_{CO_2}, and dead-space ventilation) in 6 hypercapnic COPD patients with exercise oxygen desaturation.[26] They found that, although gas exchange response to exercise was abnormal (no change in these variables), $\dot{V}/\dot{Q}$ matching *did not* worsen. Thus, sleep desaturation probably results from normal or lower demand coupled with decreased supply, whereas exercise desaturation probably results from increased demand outstripping increased or fixed supply. Cardiac output and increased tissue consumption probably play little role in sleep-associated NOD, whereas these factors play a major role in exercise desaturation.

The Role of Recurrent Desaturations on Clinical Course

P_{pa} during sleep has been observed using flow-directed pulmonary artery catheters in NOD-COPD patients during both rest and exercise.[27-34] Douglas[27] observed prolonged elevation of pulmonary artery pressure during a REM-related desaturation in a single patient. Boysen and co-workers[31] monitored four patients with Swan Ganz catheters during sleep. Transient increases in P_{pa} were regularly associated with falls in S_{aO_2} during the REM-related NOD; these transient elevations in P_{pa} were ameliorated with oxygen.

In part because of the well-demonstrated elevation in P_{pa} during desaturation, NOD has been hypothesized to be a cause or contributor to the pulmonary hypertension that occur in patients with COPD.[35,36] This, coupled with the fact that supplemental oxygen used during sleep can easily correct both the desaturation and the P_{pa} elevation,[11,27,28,37-39] suggests that long-term nocturnally administered oxygen could prevent or improve pulmonary hypertension. Because patients with a resting daytime $P_{aO_2} < 55$ torr (similar to those subjects addressed in the NOTT and MRC studies are already candidates for therapy, continuous home oxygen will presumably treat the NOD effectively. Further studies of prevalence, pathogenesis, natural history, and treatment need to be directed toward those patients who would not otherwise qualify for home supplemental oxygen: those with a $P_{aO_2} > 55$-60 torr but with REM-sleep NOD.

It is difficult to speculate what the role of exercise desaturation is in the progression of cardiopulmonary hemodynamics because data relating transient desaturation to chronic P_{pa} are scant. In general, P_{pa}, which is normal in COPD patients at rest, becomes elevated during exercise. Supplemental oxygen may ameliorate this increase in exercise P_{pa} without much obvious effect upon S_{aO_2}.[12] Exercise limitation certainly occurs without hypoxemia. Exertional dyspnea is usually associated with limitations of minute ventilation and what appears to be a maximum ventilatory threshold rather than with overt hypoxemia. Oxygen therapy during exercise frequently increases the level of maximal exercise achieved, probably by minimizing chemoreceptor stimulation associated with hypoxia. Supplemental oxygen might decrease the minute ventilation requirement of a given exercise load, thereby improving performance.[12,23]

Clinical Recognition and Diagnosis

The detection of NOD in COPD patients can be difficult and expensive. The most reliable method is to study the patient with nocturnal polysomnography in a sleep laboratory. Oximetry with an overnight recorder (or other home monitoring device) can also detect NOD. This, however, does permit potential false positives and false negatives. Artifact due to position change or pressure on the ear or finger sensor may create false positives, requiring confirmation in the sleep laboratory. Absence of oximeter desaturation does not rule out NOD because electroencephalographic monitoring is necessary to ensure that arterial saturation was measured during REM sleep. Measures of lung mechanics and gas exchange made during the day may be helpful in screening subjects but do not reliably predict patients with NOD.[39] Several investigators have tried to correlate symptoms, age, body weight, sex, pulmonary function studies, blood gases, ventilatory control, and other waking variables with NOD in an attempt to identify patients likely to have desaturation. At most, it can be assumed that the lower the FEV_1 and the awake P_{aO_2}, the more likely the patient is to show NOD. However, these parameters are not predictive in individual patients and the correlation with pulmonary function is generally poor. It has been stated that the bronchitic or type B COPD patient is more likely to desaturate than the eucapnic and normoxic 'pink puffer,'[32] but we have seen clear-cut examples of 'clinical' pink-puffer type patients with classic REM-sleep NOD.

Just as with sleep desaturation, exercise desaturation correlates in general with level of pulmonary dysfunction but poorly on an individual basis. Minh et al[40] pointed out that exercise desaturation correlates poorly with P_{aO_2}, venous admixture ($\dot{Q}_s/\dot{Q}_t$), and several other parameters of resting pulmonary function, but correlates well with deterioration of $\dot{Q}_s/\dot{Q}_t$ during exercise. Owens et al indicate that diffusing capacity (D_{LCO}) and FEV_1 are predictive of desaturation, with diffusing capacity being more specific.[41] A diffusing capacity > 55% of predicted had a specificity of 100% and a sensitivity of 68% for excluding exercise desaturation.[41]

Recent Prospective Studies on NOD Patients

Recent attempts at better characterization of COPD patients with NOD have focused on hemodynamic differences between NOD and non-NOD subjects. We have prospectively examined gas exchange, pulmonary function, red cell mass, radionuclide-gated cardiac ejection fraction, and supine cardiopulmonary hemodynamics in 36 patients with COPD.[33] All subjects had a daytime $P_{aO_2} > 60$ torr with documented REM-sleep-related NOD for > 5 minutes to a saturation ≤ 85%. The data from the NOD subjects were compared to those from 13 subjects with similar symptoms and pulmonary function but without evidence of NOD. The two groups did not vary with respect to age, spirometry, lung volumes, or D_{LCO}. The NOD subjects had a significantly lower resting daytime P_{aO_2} (70.0 vs 82.4 torr), higher alveolar-to-arterial oxygen gradient (28.1 vs 19.1 torr), and a higher hemoglobin and red blood cell mass/kg. At baseline, mean pulmonary artery pressure ($\overline{P}_{pa}$) and pulmonary vascular resistance (PVR) were higher in NOD than in non-NOD subjects (23.3 vs 20.4 mm Hg, $p < 0.05$, and 172 vs 102 dyn · cm^{-5} · s, $p < 0.0001$, respectively). Changes in $\overline{P}_{pa}$ and PVR during induced hypoxia and hyperoxia revealed no obvious difference in pulmonary vascular reactivity between groups.

In addition to resting hemodynamics, we used exercise to stress the pulmonary circulation of a subset of patients selected from the described group because of their ability to sustain steady state exercise for a period of 7 minutes.[34] The purpose was to see whether exercise amplified underlying cardiopulmonary-hemodynamic differences between NOD and non-NOD subjects. Upright exercise cardiopulmonary hemodynamics including P_{pa}, PVR, and pulmonary capillary wedge pressure (P_{cw}) were measured using an indwelling right-heart catheter, while arterial blood and expiratory gases were collected to assess metabolic and other gas exchange parameters (Fig. 3). Both groups showed increase in P_{pa} during exercise, but the increases in systolic and $\overline{P}_{pa}$ and driving pressures ($P_{pa} - P_{cw}$) were disparately higher in NOD subjects. Exercise desaturation was not predictive of NOD. The presence of NOD was predictive of a resting PVR greater than 120 dyn · cm^{-5} · s, whereas mean S_{aO_2} during sleep and daytime P_{aO_2} were unable to separate COPD patients above and below this level. This indicated that pulmonary vascular-hemodynamic abnormalities could be better explained by the presence of nocturnal episodic desaturation than by mean nocturnal S_{aO_2} or differences in resting daytime P_{aO_2} between NOD and non-NOD subjects. The P_{aCO_2} increased during exercise in subjects with NOD by 4.7 torr ($p < 0.001$), whereas the change in the non-NOD group was not significant. Gas exchange determined by measuring ventilation, expired gases, and arterial and venous blood gas tension and S_{aO_2} was worse in the COPD patients with NOD than in those without NOD.

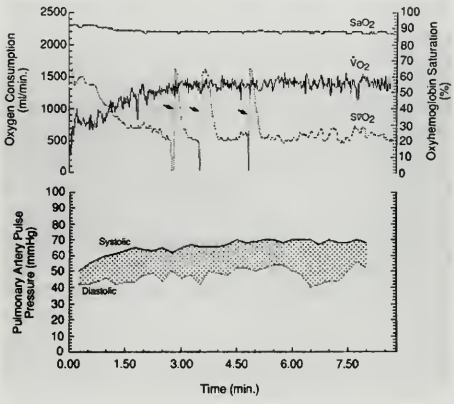

Fig. 3. Metabolic, saturation, and pulmonary artery pressure data collected during a steady state exercise test in a subject with nocturnal oxygen desaturation (NOD). Oxygen consumption ($\dot{V}_{O_2}$) reaches a plateau at about 3 minutes while oxygen saturation (S_{aO_2}) falls from 91 to 88% and mixed venous oxyhemoglobin saturation ($S_{\bar{v}O_2}$) falls to approximately 22% by the third minute and remains stable. Artifactual increase in $S_{\bar{v}O_2}$ resulting from inflation of the balloon and wedging of the catheter tip is designated by arrows. The slow increase in pulmonary artery pressure is pathologic. (From Reference 34, with permission.)

Not only is it difficult to determine who the NOD patients with COPD are without polysomnography, it is poorly understood who among non-NOD COPD patients will go on to become desaturaters as their disease progresses. We have examined the natural history of non-NOD COPD patients by nocturnal polysomnography over time to determine how many and which patients would go on to develop NOD.[42] Thirty one clinically stable COPD patients

with a $P_{aO_2} \geq 60$ torr were polysomnographically studied at baseline and at a mean time of 42.5 months after baseline study. Eight of the subjects developed REM-sleep NOD on follow-up (Fig. 4), whereas the other 23 subjects showed no evidence of REM-sleep-related NOD. The appearance of REM-sleep NOD was only minimally related to initial P_{aO_2}, P_{aCO_2}, and mean nocturnal S_{aO_2}. At follow-up, the onset of NOD was always associated with deterioration of daytime P_{aO_2} and P_{aCO_2}, and occurred mainly in those patients with the most severe baseline derangement of spirometry (lung mechanics). On the other hand, one group (Group IIA) showed equivalent deterioration in daytime P_{aO_2}, a stable P_{aCO_2}, but less severely deranged baseline mechanics and a fall in mean nocturnal S_{aO_2} without onset of episodic NOD (Fig. 4). The findings in this latter group suggest that the onset of NOD is not purely a result of decreasing daytime P_{aO_2} because these patients showed clear-cut deterioration of daytime P_{aO_2} and mean nocturnal S_{aO_2} but no sign of REM-sleep desaturation.[42] The onset of REM-sleep NOD is mainly related to a severe derangement of lung mechanics with deterioration of resting awake gas exchange (progressive hypoxemia, hypercarbia, and worsening airflow). The onset of REM-sleep NOD over time in some patients with a P_{aO_2} above 60 torr is a sign of progression of the underlying disease process.

A central question in the dilemma facing clinicians about whether to prescribe nocturnal home oxygen for NOD during sleep is, Does nocturnal oxygen cause sustained reduction in pulmonary artery pressure, thereby potentially preventing progression of pulmonary hypertension and reducing morbidity of COPD in the face of NOD? We attempted to study this question.[43] Fifty-one patients with moderate-to-severe COPD and a daytime P_{aO_2} > 60 torr were followed for 3 years: 38 had proven REM-sleep NOD and 13 had no desaturation. The 38 NOD subjects were randomized to nasal oxygen or room air during sleep, in a double-blind fashion. Nineteen subjects initially received nocturnal oxygen at 3 L/min delivered by nasal cannula from bedside oxygen concentrators and 19 received room air at 3 L/min also delivered via nasal cannula from identical concentrators rendered defective by the respiratory therapist. The output of all concentrators was checked monthly to assure appropriate function.

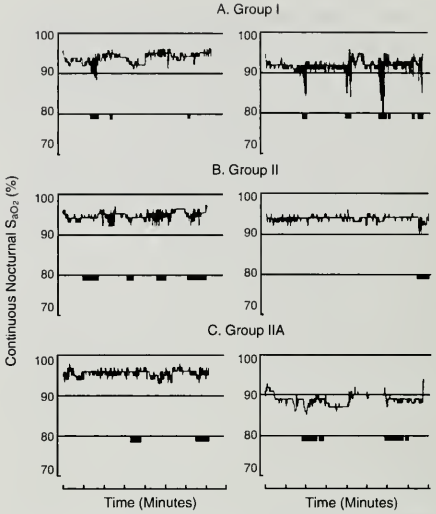

Fig. 4. Continuous nocturnal S_{aO_2} recordings (baseline left, follow-up right) in 3 patients with COPD representing each of 3 patterns of evolution of nocturnal S_{aO_2} over approximately 3 years. A. Group-I subjects show little or no evidence of NOD during initial studies despite the presence of REM sleep, but follow-up polysomnography later reveals that REM sleep is accompanied by oxyhemoglobin desaturation to levels as low as 80%. B. Group-II subjects without NOD at baseline show, on subsequent studies, that S_{aO_2} remains similar to or slightly below baseline without the appearance of REM NOD. C. Group IIA represents a subgroup (n = 8) of Group-II patients who show a fall in daytime awake P_{aO2} during follow-up, a 4% or more fall in mean nocturnal S_{aO_2}, but no REM-sleep NOD. (From Reference 42, with permission.)

The NOD subjects who received oxygen during sleep for 36 months showed a significant downward trend in P_{pa} with an overall fall of 3.7 mm Hg, compared to NOD patients treated with room air whose P_{pa} increased by 3.9 mm Hg ($p < 0.02$) (Fig. 5). PVR tended to increase in all groups, but none of the changes were significant over time or between groups. It is important to note that there was a 3.1-mm-Hg-rise in P_{cw} in the sham group, and a 3.3-mm-Hg fall in P_{cw} in the oxygen-treated group, the trends being significantly different at $p < 0.03$. There was also a small (but nonsignificant) decrease in resting radionuclide left ventricular ejection fraction in both groups. These findings suggest that the

improvement in oxygen-treated subjects may, at least in part, be related to some effect of oxygen on left ventricular function rather than on the pulmonary circulation. Nonvascular parameters of hypoxia such as hemoglobin and red blood cell mass did not differ between the sham- and oxygen-treated groups. An important problem with this study was a very high dropout rate mostly due to death or development of persistent daytime hypoxemia ($P_{aO_2} < 55$ torr) requiring removal from the study. Mortality was decidedly higher in the desaturating patients compared to nondesaturaters, but no significant survival difference was seen between oxygen and sham-treated desaturaters. This study showed that nasal supplemental oxygen used during sleep to reverse episodic NOD in COPD patients has a beneficial effect in reducing P_{pa}.[43]

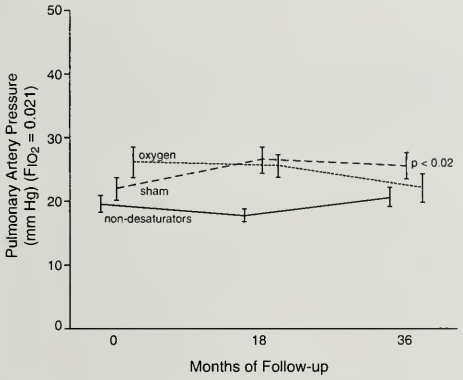

Fig. 5. Evolution of mean pulmonary artery pressure (measured with the subject supine) at 0, 18, and 36 months in all subjects completing the study. The solid line represents nondesaturating patients (n = 13) not included in the group comparisons, showing an approximate 1.4-mm-Hg increase in mean pulmonary artery pressure over 36 months. The dotted line represents desaturating patients treated with supplemental oxygen (n = 7) and shows a 3.7-mm-Hg fall in mean pulmonary artery pressure over 3 years. The broken line represents desaturating subjects treated with compressed air (n = 9), showing a 3.9-mm-Hg fall in mean pulmonary artery pressure over 3 years. The difference in change of pulmonary artery pressure between study groups (sham vs oxygen) is significant at $p < 0.02$ using two-way ANOVA with repeated measures. The error bars represent ± 1 SEM (standard error of the mean). (From Reference 43, with permission.)

Mortality and Survival in NOD Subjects

No prospective studies have examined mortality in NOD subjects. However, a recent multicenter study examined survival in a retrospective fashion in these patients.[44] Survival was examined in 169 COPD subjects having baseline polysomnography, spirometry, and blood gases performed between January 1980 and January 1990. Survival was determined as of June, 1990. Because two published definitions of nocturnal desaturation existed, both were used to classify subjects as NOD and non-NOD. One is the previously discussed definition considering *episodic* desaturation associated mainly with REM sleep (fall in saturation for 5 minutes or more to or below 85%, Definition I) and one considering > 30% of time in bed spent with a saturation of < 90% (Definition II) to be significant. Median follow-up time was 3.4 years. By both definitions, survival corrected for age was significantly better in non-NOD subjects. However, when stratified for supplemental oxygen use (patients who had received any type of supplemental oxygen eliminated from the analysis), survival remained better only in subjects separated by Definition I (Fig. 6).[44] There was a trend toward increased survival in 35 oxygen-treated vs 38 non-oxygen-treated NOD subjects (Definition I), but this difference was not statistically significant. Survival was independently related to age, spirometric values, and classification as NOD or non-NOD but not to daytime blood gases.

The Need for a Study of Supplemental Oxygen for NOD Patients

The current guidelines for the administration of continuous home supplemental oxygen in patients with COPD are based upon successful demonstration of prolongation of life in two well-controlled prospective studies.[1,2] As stated previously, these guidelines are aimed at a population that will already have demonstrable pulmonary hypertension, which, when recognized after the onset of right heart failure, portends a poor prognosis.

Varying with geographic region, in our experience, the rental cost of a home oxygen concentrator with minimal backup equipment may cost from $2,400 to $4,000/year/patient. We know from previous work[34] that if there are 16 million patients in the

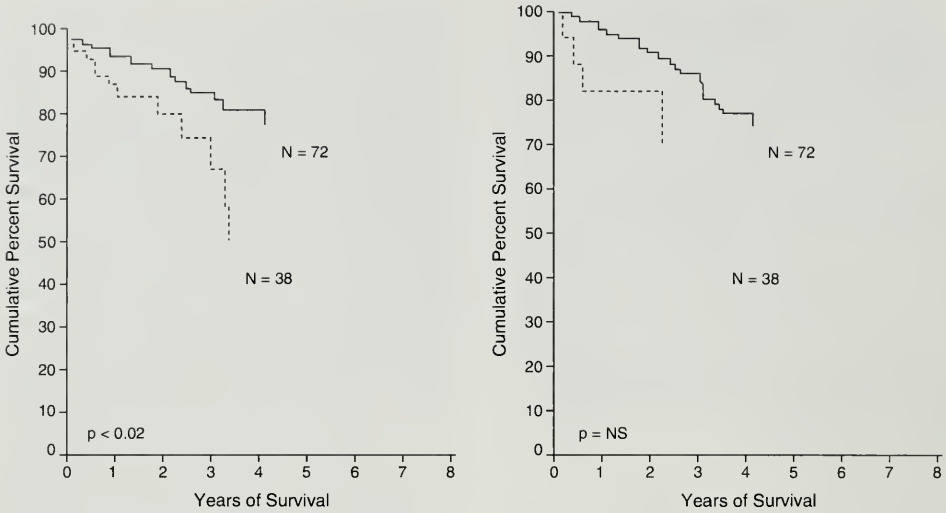

Fig. 6. Actuarial survival estimates on NOD (broken lines) vs non-NOD subjects (solid lines) after oxygen-treated subjects were removed from comparison. On the left, patients separated into non-NOD and NOD groups based upon the episodic occurrence of desaturation (from a baseline sleep S_{aO_2} of 90% for 5 minutes or more to or below 85%, Definition I) showed significantly better survival. On the right, patients separated into non-NOD and NOD groups based upon a nonepisodic definition of more than 30% of time in bed spent below an S_{aO_2} of 90%, Definition II. Survival was not different between groups by this definition. (From Reference 44, with permission.)

U.S. with COPD, and 25% have some element of NOD, the potential market for home supplemental oxygen could be as high as 4.0 million patients with a cost of billions of dollars to individuals, government, and third-party insurance carriers. An ACCP-NHLBI national conference on oxygen therapy[45] has recommended oxygen therapy during sleep for patients "who develop P_{aO_2} values of 55 torr or less during sleep which is associated with hypoxic organ dysfunction." HCFA (the Health Care Financing Administration) currently reimburses for nonhypoxemic COPD patients who have a 10% fall in oxygen saturation during sleep. Despite this, at present little evidence indicates that supplemental oxygen is beneficial to this group.

Increased mortality among patients with NOD and daytime $P_{aO_2} \geq 60$ torr has been demonstrated retrospectively. However, no well-controlled prospective study has shown that nocturnal supplemental oxygen alters mortality or clinical course other than slightly lowering P_{pa}.[43] Therefore, it is still ethical to randomize patients with NOD for prospective treatment with oxygen or placebo to determine the natural history and therapeutic outcome in those who receive supplemental oxygen. If in the future, the prescription of nocturnal oxygen becomes widely accepted in patients with NOD as has been recommended, a properly controlled prospective study might not be possible. Because cor pulmonale occurs late in lung disease and may shorten life, its prevention with early oxygen supplementation might significantly reduce mortality and morbidity.

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Fletcher Discussion

Celli: I admire your work in the sleep area, but I am a little worried that you may have chosen the wrong marker—pulmonary hypertension—if you look at the work of Levi-Valensi et al,[1] your own studies,[2] and the NOTT autopsy study[3] that showed that you couldn't distinguish between responders and nonresponders in terms of the autopsy finding in the vessels. On the other hand, if you look only at survival, you, again, may be undertreating other things that I think you ought to look at—perhaps, quality of life and daytime performance. I don't see cardiologists only looking at strokes when they treat hypertension. They are also looking at quality of life. Last, if you are going to look at your patients, I think part of the problem is whether we can correctly identify significant desaturation. You must come up with a definition of desaturation that screens out those patients who may not need oxygen and includes those who may need oxygen. I don't know where to place the ceiling or whether your data would allow you to go back and say, "OK, it's not the patients who have P_{O_2}s in the 70s but maybe in the lower 60s." If you narrowed that group, I think that it might be of some value to put that group on oxygen. Second, I would like to give some opinion on exercise. There are enough data to show that exercise not only decreases dyspnea but also postpones respiratory muscle fatigue and shifts work away from accessory muscles to the diaphragm. The reason why I say that is that a lot of subjects who have severe COPD, even if they do not desaturate, have very short exercise times. If we give oxygen to such people (those who can only exercise for 2 minutes or so)—although no outcome measures have been done until now—we can prolong their exercise time, we therefore may be able to train those muscles better. Because this will be published I believe that something should be said about the possibility that this could be one form of therapy that might enhance training.

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Fletcher: Do you want me to respond to that?

Celli: To the former.

Fletcher: First of all, there is no doubt that exercise duration and work level can improve with supplemental oxygen in patients who desaturate. Those are old data. There are also pre-1980 data in patients who do not desaturate to suggest that supplemental oxygen can also improve duration and exercise level.[1] That's somewhat controversial. To my knowledge, there are no data to show that survival or pulmonary pressure is affected long term by supplementing oxygen in nondesaturaters. I don't think it's controversial that you ought to supply a patient who desaturates during exercise with portable oxygen, ambulatory oxygen, if you can prove that his exercise tolerance improves with supplemental oxygen. I don't even think chronic outcome is questioned. With regard to sleep, I don't know how to come up with a definition of what's hemodynamically significant and what's not. With regard to outcome, I certainly wouldn't skip quality of life, number of days of hospitalization, perhaps psychometric improvement, but those are the parameters that have wide standard deviation and require large cohorts and control groups. I think that you will never sell a cooperative study to look at this question if you don't have *the* major end point—survival, now that we know (I think we know) that survival in desaturaters is decreased. In fact, you can calculate, and I've done it, that we would need 273 patients followed over a 3-year period, in a double-blind, prospective study with supplemental versus sham to determine survival difference. I think in such a study you should incorporate such parameters as noninvasive cardiopulmonary function, hospitalization days, quality of life, whatever you want to define that as, and maybe psychometric results. I think you should look at all of those things. I certainly wouldn't advocate just looking at survival. But I don't think you'll sell the study unless you do look at survival.

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Hill: Your serial data show that nondesaturaters sometimes became desaturaters,[1] so how stable are these populations? Are some of the desaturaters nondesaturaters a month later? Are they shifting back and forth?

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Fletcher: Yes, they move in and out of the group initially. One of the things that we did over the 3-year period was to restudy them every 18 months to make sure they remained desaturaters. These people did, but the reason they did is because we screened them many times before we put them in the study. What we did find was a small number of patients who were desaturaters at their initial study who then subsequently became nondesaturaters. What it boiled down to (and I think this is very closely related to the NOTT data[1] and Levi-Valensi's data[2]) was that at 3 months you can drop about 30% of patients who have a P_{aO_2} below 55 at your initial gas measurement but who go above that level at the end of 3 months. When we took patients who were within 2, 3, or 6 weeks of an acute exacerbation and studied them in a sleep lab, we found that they were desaturaters; then we waited a few more weeks, brought them back, studied them again, and found out they were not.[3] I think that acute exacerbation can be associated with the onset of nocturnal desaturation, and the farther away the patient is from the exacerbation, the more likely he is to have adequate saturation. But, definitely, there are a fixed number of desaturaters, and this 25% figure is taking into account repeated studies to eliminate those who are going to be desaturaters only for a short period of time—2 or 3 months.

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Make: Third-party payers have their own rules for reimbursement that are not always consistent with state-of-the-art care. The Durable Medical Equipment Regional Carriers (DMERC) are rewriting Medicare criteria for oxygen therapy, which the AARC, the ATS, and the ACCP have all commented upon. The initial Medicare policy stated that if the P_{O_2} drops more than 10 mm Hg during sleep—for example from 90 to 80—that would qualify you for nocturnal oxygen. We realize that's not appropriate, and we're trying to get that changed.

Decker: In the beginning, we talked about FRCs dropping during REM sleep, resulting in V-Q mismatching. I wonder whether noninvasive ventilation might be an alternative mode of therapy for this group of patients? If you're trying to increase their ventilation, increase their P_{O_2}, can you do it with noninvasive ventilation, rest their diaphragm, and have a better outcome?

Fletcher: That depends on what you mean by noninvasive ventilation.

Decker: Bilevel positive pressure, or BiPAP™, at night.

Fletcher: Let me address these issues one at a time. I have seen several studies looking at nasal CPAP,[1,2] and, no, that does not reverse the desaturation, even though it theoretically should raise FRC. I know of no studies that have systematically or prospectively looked at bilevel positive pressure. Theoretically, if you prevent the fall in saturation and you try to maintain the tidal volume at the same level prior to REM-sleep onset, you should improve saturation. Now, what I would ask you is, how much cheaper is it to provide bilevel positive pressure and what is the compliance level of bilevel positive pressure versus nasal oxygen? I find that in apnea patients, compliance with CPAP and, to some extent, bilevel positive pressure, is poor. For CPAP it's terrible. The patients tell you they use it, and you look at them and wonder. The recent study from the University of Pennsylvania group,[3] published in the *American Review of Respiratory Disease*, demonstrates that quite well. Of 31 patients, only 2 patients used their nasal CPAP 5 nights a week for 7 hours per night, despite severe apnea. My concern is I'm afraid that patients with COPD (who don't like masks to begin with) aren't going to comply; so, it might be a moot point. They would comply better with nasal oxygen than with a machine that requires a mask on their face.

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Bach: I won't argue with what you're saying about patients who really aren't grossly underventilating. I think it's reasonable, but I think we have to set guidelines for oxygen therapy for this population, and perhaps for the central apnea patients as well. But what I'm afraid of is that this is going to be applied to neuromuscular patients. Smith[1] proposed that Duchenne muscular dystrophy patients be treated with oxygen therapy, and I've seen that polio patients with severe restrictive pulmonary syndromes and hypercapnia are treated with oxygen all over the United States. I don't know if the HCFA oxygen-use guidelines specifically concern the COPD population, but I think they should. We have to be very careful not to apply them to the neuromuscular population. One of the reasons I say that is not only because of your comment, Mike (Decker)—that you can correct the oxygenation problem by providing noninvasive ventilation for these patients, but also our preliminary studies suggest that patients with neuromuscular conditions who are treated with oxygen have a much higher incidence of pulmonary complications before requiring ventilator use than if they're not treated at all.

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Fletcher: What you're saying is that regardless of the ability to oxygenate them at night, they do worse if they're oxygenated with oxygen versus with a ventilator.

Bach: There's no question about that. The patient with global alveolar hypoventilation should not be treated with oxygen. We should manage the ventilatory impairment to help the patient.

Fletcher: What about the compliance with—I presume you're speaking of BiPAP™?

Bach: No. I don't use BiPAP™ much for this population, but I use portable ventilators, portable pressure and volume-triggered ventilators. I use a nosepiece or a mouthpiece for daytime ventilatory support and a mouthpiece with lipseal for support during sleep. I give the patient the choice, but I monitor the nocturnal saturations and demonstrate a significant improvement with treatment. When monitored, I also see an improvement in their CO_2. I've had patients whose mean nocturnal saturations were 55% or less, who with just nasal ventilation corrected to 95%. All of their symptoms dissipated and they are fine. But such a patient treated with oxygen does not improve. You may be a little less dyspneic, but you'd have more hypersomnolence, more headaches, more nightmares, and more problems with concentration

and greater risk of intercurrent pneumonia and acute respiratory failure. So, it's very important not to extrapolate these oxygen-use guidelines to patients with global hypoventilation.

Celli: Should we have every COPD patient studied with overnight oximetry? If not, whom should we study?

Fletcher: Precisely the point I'm trying to make is that my data do *not* support that. All I'm saying is that in a small group of patients, I was able, to my satisfaction, to demonstrate a decrease in pulmonary artery pressure, which I might add was made up of, in great part, a decrease in pulmonary capillary wedge pressure. So, I'm not even sure that correcting pulmonary hypertension, whether you call it muscular hypertrophy or pulmonary artery contraction as a result of hypoxemia, was the basic cause of the improvement in his pulmonary artery pressure. The message I'm trying to get across is that we don't have the data to proceed with mass screening of patients with COPD to look for nocturnal desaturation because we don't have the data that nocturnal supplemental oxygen will improve survival or quality of life. I think that survival is a more definite and a better outcome to look at than pulmonary artery pressure because patients don't come in to you complaining, "My pulmonary artery pressure is elevated." They die, and that's a definite end point. We know now that mortality just by this marker of nocturnal desaturation, is higher than it is in nondesaturaters. Therefore, if we correct it (which is what HCFA would have us believe we ought to do by their new guidelines), are we really improving anything? Does the patient feel better? Are there fewer hospital days? Does he live longer? We don't have that data.

Celli: Yes, but who should we send for a study or an oximetry? That's my question to you.

Fletcher: Clearly, in my mind, the only person I send for oximetry is a COPD patient with a daytime P_{aO_2} that doesn't qualify him for oxygen but who has signs of cor pulmonale. I think that's where we should keep the state of the art until we have more data.

Hill: Before we entirely dismiss PA pressures, I have a question that has come up at a lot of our conferences where I'm from. Do patients who don't have daytime hypoxemia or hypoventilation but who do desaturate significantly at night during sleep-disordered breathing go on to develop pulmonary hypertension?

Fletcher: Oh no, I can answer that very definitely. In this 25%, which works out to be 36 patients, over 70% had pulmonary hypertension, measured at baseline, if you define it as PA pressure greater than 20 mm Hg.

Hill: Mean PA pressure?

Fletcher: Yes, 70% of them were over 20, and I think the figure was 23 or 21.7, somewhere in the 22-23 range. So, they already had elevated resting, supine pulmonary artery pressures.

Hill: Mild.

Fletcher: Mild.

Hill: And do you think, symptomatic?

Fletcher: No.

Hill: Subclinical.

Fletcher: Subclinical. We looked at EKGs. No consistent change on EKGs. We looked at the diameter of the right lower lobe pulmonary artery. It was nondiscriminatory. Nothing to really point these patients out clinically.

Make: You're suggesting that in people who have signs of hypoxia but who have normal daytime P_{O_2}, you should look for nocturnal desaturation. How large is that population?

Fletcher: I don't know the answer to that question. I would think it's rather small. Now that you've brought that up, there's a point I need to make that's very important. If you have a patient with COPD who comes in with cor pulmonale or any sign of pulmonary hypertension, a sleep study is absolutely mandatory because what you will find more often than just REM desaturation in this group is the "overlap" syndrome—significant sleep apnea and COPD. Those patients need their apnea treated very aggressively because you can really improve their daytime blood gases by improving their nocturnal desaturations from the apnea. So that's the major indication, not that you're going to anticipate finding REM desaturaters, per se. What you're going to find the majority of the time are patients with overlap syndrome. If I tried to give you a figure on that, it would be very biased because of my working in public hospitals. Such patients are extremely common because patients who've abused cigarettes frequently have poor dietary habits and abuse food also. They are overweight, have sleep apnea, and COPD—that is a lethal combination.

Pierson: We come back to cost-benefit, actually. Both the investigation and therapy of nocturnal desaturation is pretty expensive. I think you've shown us that we have no very compelling evidence that either detecting this or doing something about it makes a readily quantifiable difference that would stand up in a cost-benefit way of approaching this problem. I think that that is a very valuable process to have gone through. If you consider that we must confront

the finite nature of our resources, these things being fairly expensive means that to treat this particular situation would be a fairly expensive proposition. Until we have definite outcome-benefit data, it's probably best that we don't do that.

Fletcher: That's precisely the point. Let me expand on that comment and come out with a recommendation. My recommendation is that we not follow HCFA reimbursements, and that for the time being we look for these patients and prescribe oxygen on the basis of our expertise and facilities. For example, if we have sleep labs and if we have patients who are not hypoxemic (P_{aO_2} > 60 torr) who come in with some signs of cor pulmonale, we should obtain nocturnal sleep studies, and if they are desaturaters, we should go ahead and treat them. But, by all means, we should not screen broad populations of COPD patients. If we find desaturaters, we treat them, but the major recommendation is that we are ripe for a prospective multicenter study, similar to the NOTT study.[1] I think what needs to be stressed is that we haven't done anything prospective in large populations since the NOTT study[1] and the MRC study[2] in the 70s. With the expanding technology, as in so many specialties in medicine, our technology has outstripped our ability to treat. We've got to stop at this point and examine prospectively, with a multicenter trial, whether we should proceed with looking for these patients and treating them. We simply don't have the database to support what HCFA has suggested they're going to reimburse us for. If we abuse it by going ahead and doing this without the proof, then in the future it's going to be a rebound or a swinging pendulum in the opposite direction, and we're going to have limitations on our ability to treat patients who really do need it.

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Travel for the Technology-Dependent Individual

James K Stoller MD

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Introduction

With improvements in transportation over the last several decades, travel over long distances has become commonplace in modern life. At the same time, technologic advances now permit increased travel opportunities for technology-dependent individuals (eg, oxygen dependent and mechanical ventilator dependent), although limitations do persist. The purpose of the current paper is to review aspects of travel for the technology-dependent individual. Specifically, travel by commercial air carrier is reviewed first, including consideration of the physiologic effects of altitude exposure, a review of physiologic conditions and medical hazards associated with commercial air travel, available strategies for predicting in-flight arterial oxygen tension, and practical tips for the air traveler needing in-flight or on-ground oxygen while traveling. Attention then turns to the issues of travel with oxygen over ground and/or water and, finally, to travel with technology other than oxygen, especially with mechanical ventilation.

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Travel by Air

Effects of Acute Altitude Exposure

Exposure to altitude has protean effects that are affected by prior residence at altitude and by the chronicity of altitude exposure.[1] The acute hypobaric hypoxic exposure of commercial air travel is

short-lived (ie, generally ≤ 12 hours) and the effects are mainly characterized as pulmonary and hemodynamic, although other symptomatic effects (eg, dizziness, near-syncope, nausea) are commonly described. The effects of altitude exposure are summarized by Dalton's and Boyle's Laws, which dictate that inspired oxygen tension falls and that gas density decreases under hypobaric conditions.[2]

The pulmonary effects of acute altitude exposure relate to decreases in barometric pressure and the consequent falls in oxygen tension.[3-6] These effects include diminished inspired and arterial oxygen tensions, increased respiratory rate, and increased minute ventilation with elevation of the ventilatory equivalent ($\dot{V}_E/\dot{V}_{O_2}$).

The major hemodynamic effects of acute altitude exposure include elevation of heart rate and cardiac output, preferential redistribution of perfusion to essential organs such as heart and brain with diminished perfusion to other organs (eg, skin), and vasoconstriction of the pulmonary arteries, mediated by alveolar hypoxia.[4,7]

Besides effects related to hypoxia, other effects of altitude exposure relate to decreased gas density. For example, at an elevation of 8,000 feet, gas density is approximately 30% lower than that at sea level.[4] Gas trapped in closed spaces, such as sinuses, blocked middle ear, noncommunicating lung bullae, pleural spaces, or abdominal viscera expands and may cause a variety of symptoms including acute discomfort, pneumothorax, abdominal distension, or actual dehiscence of recent abdominal wounds.[3]

As a special case of acute hypobaric hypoxic exposure, several studies have considered the effects of acute altitude exposure in patients with chronic lung diseases, including chronic obstructive lung disease (COPD).[8-11] As described later, the altitude inside the cabin of an aircraft at cruising altitude can be considered to be 8,000 feet as a general worst-case scenario.[3,4] In an early study simulating commercial air flight by exposure to 8,000 feet ambient pressure in a hypobaric chamber, Tomashefski et al[8,9] observed adverse clinical effects in 8 of 20 patients (40%) with a variety of chronic lung diseases (eg, pneumonectomy, diffuse infiltrative disease, and emphysema). These adverse effects included intolerance to hypoxia (characterized by severe dyspnea), inability to hyperventilate, hypoventilation, and severe abdominal bloating and discomfort due to gaseous distension.

In contrast to these early reports, subsequent studies exposing patients with COPD to altitudes simulating commercial air flight, either by actual ascent to altitude or by short-term hypobaric chamber exposure, have not reported adverse clinical effects.[10,11] For example, Graham and Houston studied short-term adaptation to altitude exposure (1,920 m, or ≈ 6,000 ft) in 8 patients with COPD (mean FEV_1 = 42% predicted, range 25 to 78% predicted).[10] Except for mild fatigue at altitude, all patients tolerated the exposure well and no significant changes were observed in cardiopulmonary parameters, including minute ventilation, respiratory rate, oxygen consumption, or resting heart rate. In a more recent study, Berg et al exposed 18 COPD patients [mean (SD) FEV_1 0.97 (0.32) L, 31 (10) % predicted] to 8,000 feet altitude for 45 minutes in a hypobaric chamber, initially without and then with supplemental 100% oxygen at 4 L/min.[11] Altitude exposure and resultant hypoxemia [mean (SD) P_{aO_2} at altitude 47.4 (6) torr] was well tolerated subjectively in all patients. Mean (SD) heart rate did not increase and, although cardiac ectopy increased slightly with hypobaric hypoxia [from 37 (16) complexes/h to 45 (25) complexes/h], this was clinically benign and returned to baseline with oxygen supplementation [38 (16) complexes/h]. Neither systemic blood pressure nor pulsus paradoxus changed with altitude, although oxygen administration was associated with a decreased pulsus, which was ascribed to decreased work of breathing associated with decreased airflow resistance from lower gas density.

Overall, although altitude exposure has been associated with a number of cardiopulmonary effects, most studies considering short-term exposure of COPD patients to altitudes achieved during commercial air flight have shown excellent tolerance with minimal cardiopulmonary derangement despite significant hypoxemia.

Determinants of Cabin Altitude in Commercial Aircraft

Ascent to altitude causes a decline in barometric pressure and a concomitant fall in the partial pressure of inspired oxygen. Specifically, inspired oxygen tension (P_{IO_2}) is determined by the equation

$$P_{IO_2} = 0.21 \times (P_B - 47),$$

where P_B is atmospheric barometric pressure and 47 torr is the partial pressure of water vapor at body temperature.

Thus, if P_B at 8,000 feet is 568 torr, P_{IO_2} at 8,000 feet is 108 torr. As a rule of thumb, P_{IO_2} falls by approximately 4-5 torr/1,000 feet of elevation.[12]

Although commercial airliners may cruise at altitudes up to 40,000 feet (where the ambient barometric pressure is 141 torr and P_{IO_2} is 29 torr), pressure within the aircraft cabin (so-called cabin altitude, CA) is kept higher than pressures outside the aircraft by pressurizing the aircraft cabin. Specifically, the aircraft maintains a constant pressure gradient between cabin pressure and outside ambient pressure. The maximal achievable gradient between ambient pressure and CA is a feature of the type of airplane and varies among commercial aircraft (Table 1), such that higher maximal gradients in more modern airplanes (eg, 8.3-10.7 psi) permit flight at higher altitudes.[12,13] Maximal aircraft flight altitude is limited by Federal Aviation Administration (FAA) requirements, including that the cabin altitude must be kept below 8,000 feet during usual flight. An allowance for cabin altitudes up to 10,000 feet is made for temporary diversions to avoid adverse weather conditions.

Table 1. Aircraft Cabin Pressurization*

Aircraft Type†	Differential Pressure (psi)	Cabin Altitude (ft)‡
B-727	8.6	5,400
B-757	8.6	5,400
B-767	8.6	5,400
B-747	8.9	4,700
B-737	7.45	8,000
DC-8	8.77	5,000
DC-9	7.76	7,300
DC-10	8.6	5,400
A-300	8.25	6,100
A-320	8.3	6,000
L-1011	8.4	5,800
BAC-111	7.5	7,900
Concorde	10.7	1,000

*Adapted from Reference 12, with permission.
†n = 204 flights on 28 commercial airlines with 16 different aircraft.
‡Cabin altitude higher with new aircraft (mean 7,004 vs 5,820 ft, p = 0.002).

Cottrell[12] measured cabin altitude in 204 commercial aircraft and showed that as a result of higher pressurization capabilities, more modern aircraft (eg, 767, A-310) fly higher and expose passengers to higher cabin altitudes, although still below the 8,000-foot FAA ceiling. Also, Aldrete and Aldrete[14] directly measured ambient oxygen percentages during 22 commercial flights on 707 or DC-9 aircraft. During flights between 29,000 and 37,000 feet, equivalent oxygen percent (normalized to sea level) was 15.2%.

Medical Hazards Associated with Commercial Air Travel

Given the large number and health diversity of passengers using commercial air travel, adverse medical events during commercial air travel are expected. For example, Gong has estimated that 5% of commercial air travelers are under medical care.[4] Surprisingly, however, the frequency of adverse medical events during commercial travel is low. As reviewed in Table 2, five studies have catalogued the frequency of medical hazards during commercial air travel.[15-19]

Cummins et al[15] conducted a prospective survey of travel through the Seattle-Tacoma airport for 1 year beginning September 1, 1986. Among 14.4 million passengers on 274,000 commercial flights, 754 travelers were evaluated for medical complaints either during travel or while passing through the airport. Of these 754 travelers, 564 (75%) experienced difficulty in the airport, whereas the remaining 190 (25%) experienced problems in-flight. Of these 190 travelers, 7 developed in-flight medical problems requiring unscheduled aircraft landings and 5 persons experienced cardiac arrest. Of the 754 travelers evaluated, most experienced medical problems (60%), of which gastrointestinal and cardiac problems were the two most specific (Table 3).[15-20]

In a second review of medical problems encountered during commercial air travel, Cottrell et al[16] reviewed the use of the on-board medical kit during travel on United Airlines over a 1-year period.[16] Among 55 million passengers and 680,000 departures, the kit was opened during flight 364 times or once per 1,900 flights and once per 150,000 passengers. Patients for whom the medical kits were opened showed a male preponderance (57%) and

Table 2. Summary of Available Reports of Medical Emergencies during Air Travel

First Author (Year)	Site or Source	Number of Travelers	Frequency of Emergencies (n)	
			All Travelers	In Flight
Cummins (1988)[15]	IATA Survey	245.3 million	1/3,200,000 deaths	—
Cummins (1989)[19]	Sea-Tac	14.4 million (274,000 flights)	1/19,000 (754)*	1/39,600 (190)
Speizer (1989)[17]	Los Angles International Airport	8.74 million	—	1/33,000 (260)
Skjenna (1991)[18]	Air Canada	13,353,000	3.4/100,000†	—

*Unscheduled landings in 7/190; 5 cardiac arrests of which only 1 occurred on board plane.
†Total 2 deaths.

mean (SD) age of 54 (16) years. The most common medical problems encountered during flight were syncope/near-syncope (29% of problems), cardiac-related problems or chest pain (16%), and asthma, COPD, or dyspnea (10.1%). Five deaths occurred, three during flight (all of which were cardiac-related) and two during post-flight hospitalizations.

A third review of the medical problems encountered in flight was conducted by Speizer et al at Los Angeles International Airport for 1 year beginning in October 1985.[17] As in the other series, the frequency of in-flight symptoms was low (260 out of 8,735,000 passengers, or 0.003%). Twenty-five patients were hospitalized (1/350,000) and 7 patients died (1/1.3 million), 6 related to cardiac arrest and 1 to stroke. Of the nonfatal in-flight symptoms recorded, loss of consciousness was most common (19% of complaints), followed by nausea or vomiting (17%), chest pain (13%), abdominal pain (9.5%), and shortness of breath (7.5%).

A fourth review of in-flight medical incidents on Air Canada between 1982 and 1988 was reported by Skjenna et al.[18] The rate of death was consistently low over this interval (eg, 2/13,553,000 in 1988), but total medical incidents increased slightly (from 1.7/100,000 passengers in 1982 to nearly 4/100,000 passengers in 1988) as did the number of times the in-flight medical kit was opened (128/year in 1982 vs 167/year in 1988). As shown in Table 3, cardiovascular incidents were most common (51% in 1988), followed by gastrointestinal (12%) and respiratory (10%) incidents.

Finally, in a review of in-flight deaths, Cummin and Schubach[19] reviewed records from the International Air Transport Association (IATA) that were based on reports submitted between 1977 and 1984 from 42 commercial air carriers. A total of 577 in-flight deaths were reported (72 deaths/year or 0.31 deaths/million passengers). Men were more frequently represented (66%). Notably, 77% of those patients dying reported no health problems before air travel. Cardiac causes accounted for 56% of the deaths.

Based on this experience of air travel-associated medical hazards, some airlines now screen clients

Table 3. Types of Medical Problems Encountered during Air Travel: Summary of Available Series

First Author (Date)	n Events	Types of Medical Events (n)					
		Syncope/Near	Cardiac	Asthma/COPD	Seizures	Diabetes	GI
Cummins (1988)[15]	577 deaths	—	56%	6%	—	—	—
Cummins (1989)[19]	754	4%	11%	7%	4%	2%	12%
Speizer (1989)[17]	260	9%	17%	12%	5%	—	11%
Cottrell (1989)[16]	362	29%	19%	10%	4%	3%	—
Hordinsky (1989)[20]	1,016 events 13 deaths	23%	14% 61%	2%	4%	2%	11%
Skjenna (1991)[18]	464	—	51%	10%	7%	—	12%

for prior medical conditions and refer patients to a medical evaluation service. In a recent report describing such a strategy, Gong et al reported the experience from Delta Airlines for the year 1991.[5] Of 42,770,468 passengers on Delta's domestic flights in 1991, 1,115 were referred to a medical evaluation service when a possible medical condition was noted at the time airline reservations were made. Of the 1,115 medically impaired patients, 80% (892) were referred for oxygen use during flight. Chronic obstructive pulmonary disease accounted for 44.3% of requests for in-flight oxygen, and cardiac-related needs accounted for an additional 34.1%. Another 4.6% of the 1,115 patients were pulmonary patients not needing oxygen and the remaining 15.4% (172/1,115 patients) had a nonpulmonary need mostly based on neuropsychiatric (96/172 or 55.8%), orthopedic (37/172 or 21.5%), or cardiac problems (17/172 or 9.8%). Notably, 35% of these 1,115 patients had been hospitalized within the 2 weeks before arranging air travel, suggesting that this segment of the traveling population was potentially unstable. Also, oxygen was underused during air travel; of the passengers requiring oxygen, 72.8% had flown before but only 25.1% reported using oxygen on the earlier flight.

That the outcome of preflight medical evaluation can be successful is suggested by the absence of medically significant in-flight events for the patients cleared to travel in this report. Of the 1,115 referrals, 90% (1,011) were cleared and 104 were denied travel, either based on an unstable condition or the need for medical services that could not be provided on Delta Airlines (eg, intravenous therapy and stretchers).

In summary, medical illnesses during air travel are infrequent but most commonly are due to cardiopulmonary disorders. Deaths are uncommon among travelers (including those with recent hospitalizations), and available experience suggests that the risk of medical mishaps may even be made lower with simple preflight medical screening.

Predicting In-Flight P_{aO_2}

Because supplemental oxygen is recommended when in-flight P_{aO_2} is $\leq$ 50 torr, predicting in-flight P_{aO_2} is considered worthwhile and has been studied.[3,4,6,10,21-26] Several strategies have been undertaken to predict in-flight arterial oxygen tension, including (1) administering hypoxic mixtures to measure P_{aO_2} directly at altitudes simulating the highest cabin altitude during commercial air flight; (2) measuring P_{aO_2} during hypobaric conditions, either during actual air flight or during ascent to altitude; and (3) employing prediction equations derived from studies using hypoxic inhalation as in (1) or hypobaric hypoxia as in (2).

Administering hypoxic mixtures to estimate in-flight P_{aO_2} has been undertaken in two studies and has been called the hypoxia-altitude simulation test.[3,6,22] Specifically, Gong et al[6] studied 22 patients with stable chronic obstructive pulmonary disease [mean (SD) FEV_1 44 (17) % predicted; P_{aO_2} 68 (8) torr; P_{aCO_2} 40 (4) torr] and administered three hypoxic mixtures (17.1%, 15.1%, and 13.9% oxygen) to simulate cabin altitudes of 5,000, 8,000, and 10,000 feet, respectively. Arterial blood gas measurements were obtained once a steady state had been achieved, followed by administering supplemental oxygen (100% oxygen at 1 L/min) as a bleed-in to supplement the hypoxic mixture. P_{aO_2} fell to 48 (1.1) torr at 8,000 feet (the equivalent of 15.1% oxygen at sea level) and was restored to 84 (3.9) torr with concomitant bleed-in of 100% oxygen at 1 L/min. A stepwise discriminant function analysis showed that sea level P_{aO_2} was the only significant predictor of P_{aO_2} at altitude. The predicted P_{aO_2} at altitude is summarized in Table 4, which presents the four predictive equations derived from available studies.[6,23,25,26] Based on these data, a sea level P_{aO_2} < 72 torr predicted a $P_{aO_2} \leq 50$ torr at 8,000 feet cabin altitude in 86.7% (13/15) of subjects.[3,6]

Table 4. Equations for Predicting P_{aO_2} at Altitude*

First Author	$P_{aO_2\ altitude}$ =	r†
Henry[23]	$(20.38) - (3.0)\ CA + (0.67)\ P_{aO_2\ ground}$	0.83
Gong[6]	$22.8 - (2.74)\ Alt + (0.68)\ P_{aO_2\ ground}$	0.94
Dillard[25]	$0.453\ (P_{aO_2\ ground}) + (FEV_1\ \%\ pred) + 2.440$	0.85
Dillard[26]	$P_{aO_2\ ground} \cdot e^{[0.01731 - 0.00019)\ (FEV_1\ \%\ pred)]}$	0.92

*CA = cabin altitude (ft); $P_{aO_2\ ground} = P_{aO_2}$ at ground level; Alt = altitude in thousands of ft; $P_{aO_2\ altitude} = P_{aO_2}$ at altitude.
†Correlation between measured and predicted P_{aO_2} at altitude.

In a second study using hypoxic inhalation, Schwartz et al[22] assessed lung function and arterial blood gas values in 17 patients with stable COPD [mean (SD) FEV_1 = 0.986 (0.438) L, P_{aO_2} 68 (7.3) torr, P_{aCO_2} 40.0 (5.8) torr] while breathing 17.2% oxygen to simulate a cabin altitude of 1,650 meters (5,000 feet). P_{aO_2} on 17.2% oxygen decreased to 52.5 (5.6) torr. In a second phase of this study, the prediction of in-flight P_{aO_2} was validated by direct measurement of P_{aO_2} in 13 of subjects who flew in an unpressurized plane at 1,650 m (5,000 feet). The mean (SD) P_{aO_2} during flight in these 13 patients decreased from 68.2 (8.5) torr (ground) to 51 (9.1) torr (altitude). Furthermore, P_{aO_2} breathing 17.2% oxygen agreed with the in-flight P_{aO_2} within 10 torr for 12 of the 13 patients and within 5 torr in 6 of the 13 patients. The sea level P_{aO_2} measured 1 to 2 hours before flight correlated strongly with the in-flight P_{aO_2} ($r = 0.85$), although the sea level P_{aO_2} measured within 4 months of the flight did not ($r = 0.34$).

As in the validation phase of the study by Schwartz et al, several studies have measured arterial oxygen tensions under hypobaric conditions, either during actual flight, ascent to mountaintops, or during hypobaric exposures in a chamber.[10,23-26] Henry et al[23] measured arterial blood gases during medical evacuation flights (cabin altitudes 3,000-7,500 feet) in 201 Vietnam medical evacuees. Preflight ground-level oxygen tension was found to correlate significantly with in-flight P_{aO_2} ($r = 0.83$), as shown in Table 4.

Graham and Houston compared arterial oxygen tensions at sea level and atop Mt Washington (elevation 1,920 m) in 8 patients with COPD [mean (SD) FEV_1 42% predicted (16%); mean (SD) P_{aO_2} 66 (7.1) torr].[10] Within 3 hours of ascent to 1,920 m elevation, mean (SD) resting P_{aO_2} fell to 51.4 torr but rose slightly by the third to fourth day at altitude to 54 (5.96) torr, as patients hyperventilated with continued altitude exposure.

In an early study to simulate commercial air flight in COPD patients using a hypobaric chamber, Matthys et al[24] showed that mean P_{aO_2} fell from approximately 68 to 52 torr during 3 hours of exposure to a 2,500 m (8,000 ft) altitude.

Using a hypobaric chamber to simulate cabin altitude during commercial air flight in a more recent study, Dillard et al[25] studied 18 stable COPD patients [mean (SD) FEV_1 31 (10) % predicted, mean (SD) P_{aO_2} at sea level 72 (9) torr]. Pulmonary function tests (without prior bronchodilators) and resting room-air arterial blood gas values were obtained at sea level 1 hour before decompression, followed by hypobaric exposure to 8,000 feet for 45 minutes, after which a steady state was achieved and another arterial blood gas was obtained. Analysis of ground and altitude P_{aO_2} values using stepwise forward regression showed that the determinants of P_{aO_2} at altitude were the arterial oxygen tension at sea level and the FEV_1 (or % predicted FEV_1, Table 4). Overall, this study departs from prior research in showing that the addition of FEV_1 to the prediction model improved sea level P_{aO_2} as a significant determinant of altitude P_{aO_2}, a conclusion that is reinforced by a subsequent meta-analysis conducted by the same investigators.[26] In a meta-analysis, Dillard et al pooled data from 5 published studies involving 71 COPD patients with arterial blood gas measurements at ground level and at altitude.[26] Hypoxic exposure consisted of ascent to altitude in two studies,[10,23] hypobaric chamber exposure in two studies,[24,25] and hypoxic gas inhalation in the fifth study.[3] In pooling the results from all five studies, Dillard et al[26] again observed that both ground P_{aO_2} and FEV_1 were significant determinants of altitude P_{aO_2} (Table 4). Using the derived regression, observed and predicted altitude P_{aO_2} values agreed within 1 torr for all 5 studies.

In summary, available studies suggest that altitude P_{aO_2} can be predicted best by including values of a recent sea level P_{aO_2} and FEV_1 (or % predicted FEV_1). While current guidelines suggest prescribing in-flight oxygen (usually at 2 L/min bleed-in above the liter flowrate for oxygen used at sea level) when the predicted P_{aO_2} at 8,000 feet is $\leq$ 50 torr, several shortcomings of currently available prediction methods are noteworthy: (1) validation of available regression equations in patients other than those studied to derive the equations is needed; (2) clarification of the impact of bronchodilator and other medication use on predictive accuracy is needed; (3) validated methods to predict oxygen needs for patients with other diseases, including restrictive chest diseases (eg, interstitial lung disease and kyphoscoliosis), COPD accompanied by comorbid conditions, and COPD patients with hypercapnia are not available; (4) further clarification of

the acute risks of short-term hypobaric hypoxia is needed to ascertain whether a predicted in-flight $P_{aO_2} \leq 50$ torr is the best available indication for in-flight oxygen; and (5) further clarification about the need for in-flight oxygen for travelers not requiring home oxygen at sea level is desired, given the apparent rarity of medical mishaps during commercial air travel.

Practical Tips on Planning Air Travel for COPD Patients

In planning commercial air travel for the patient with COPD, several practical tips are noteworthy (Table 5).[6,27-29] These can be considered in four phases of arranging travel:

- Interactions with physicians,
- Interactions with the air carrier and/or travel agent,
- Interactions with the oxygen vendor, and
- Personal planning for the travel day.

Interacting with Physicians. Medical evaluation prior to air travel is appropriate to assure fitness to fly as well as to ascertain whether oxygen should be recommended during flight. Should in-flight oxygen be necessary, air carriers generally require a letter from a physician, specifying the patient's clinical condition, the oxygen flowrate, and duration of in-flight oxygen requirements. Multiple copies of this letter are necessary in case multiple segments of the trip are required. As summarized in Table 6,[28-30] exceptions to this policy include several domestic and international carriers (eg, TWA, United, KLM) who will contact the traveler's physician directly for relevant information about traveling with oxygen.

Alternatives to the physician's letter are the standard Medical Information Form (MEDIF) developed by the International Air Transport Association and required by some carriers before flight, and the Frequent Traveler's Medical Card (FREMEC),[4,6] which is issued by the participating carrier's medical department and states the traveler's fitness to travel. The FREMEC card must be reviewed periodically but obviates the need for physician clearance before each trip during the valid life of the card.

Table 5. Practical Tips for Planning Travel for the COPD Patient

Interaction with Physician
Determine whether need for in-flight oxygen exists
Get multiple copies of
letter describing condition
liter flow and duration of in-flight oxygen
medication prescriptions
Consider emergency supply of antibiotics, corticosteroids
Ascertain names of physicians en route and at destination
Interaction with Air Carrier/Travel Agent
Notify of need for oxygen ≥ 48 hours before flight
Fly nonstop if possible
less inconvenient
less expensive (especially if O_2 cost is per coupon)
Travel during business hours so vendor personnel are available
Pre-arrange motorized cart or wheelchair if layover is scheduled
Try to be seated near bathroom on plane
Call airline at least 48 hours before flight to confirm
Consider using a travel agent specializing in travel for patients with medical needs
Personal Planning for Travel Day
Arrive at least 1 1/2 hours early
must pay for oxygen on day of travel
charge is either per coupon or per canister
Bring your own nasal cannula and extra length of tubing
Pack medications in carry-on luggage
Have multiple copies of prescriptions
If oxygen is needed for layover and not already arranged, First Aid Stations may have (35% have oxygen available by informal poll)
Have cash (to pay for oxygen if needed)
Interaction with Oxygen Vendor
Favor a company that has or can arrange nationwide coverage
Arrange for oxygen during layovers (if needed)
Try to learn the type of system that will be supplied (ie, to check adapters)

Other issues to be reviewed with the physician before flying include obtaining multiple copies of prescriptions for medications, possibly obtaining an emergency supply of antibiotic and/or corticosteroids (in case of a COPD exacerbation while traveling), and ascertaining the names of physicians at layovers along the travel route and at the travel destination.

Interacting with Air Carriers and Travel Agents. Because air carriers have specific and varying policies about supplying oxygen, early and repeated communications with the air carrier are required to minimize inconvenience during travel. As summa-

Table 6. Airline Requirements for Arranging In-Flight Oxygen: United States Domestic Carriers (December 1992)*

Air Carrier	Advance Notice	Fee Structure	Oxygen Supply		Traveler's Own O_2	Special Terms
			Liter Flow	Delivery Device		
Alaska	24 h	$50/Coupon	2 to 8 L/min	Mask or cannula	On board	—
American	48 h	$50/Coupon	2 or 4 L/min	Mask or cannula	Send air freight at own expense	Aisle seat, only 1 traveler per flight
Delta	48 h	$50/Coupon	2 to 8 L/min	Mask only	Check empty as baggage	Maximal 4 cylinders/flight
Hawaiian	48 h	$40/Bottle	2 to 8 L/min	Mask only	Check empty as baggage	—
Northwest	48 h	$75/Coupon	2 to 5 L/min	Mask or canula	Check empty as baggage	Sign liability release
TWA	48 h	$50/Coupon	2 or 4 L/min	Mask only	Check empty as baggage	TWA will call MD directly
United	48 h†	$50/Coupon	—	—	Check empty as baggage	United will call MD
US Air	48 h	$40/Bottle	2 to 8 L/min	Mask or cannula	Check empty as baggage	—

*Airlines not accepting passengers with O_2: America West, Southwest, Midway, US Air Express. (From References 28 and 29, with permission.)
†Domestic flights only; 7 days notice for international flights.

rized in Tables 6 and 7, airline requirements for arranging oxygen vary both among domestic air carriers and, to a greater extent, international carriers. Significant variation exists among policies regarding whether the traveler may carry on a personal oxygen supply, the amount of advance notice required to arrange in-flight oxygen to be supplied by the carrier, the cost of in-flight oxygen, and the oxygen-delivery apparatus available. Because domestic air carriers prohibit patients' bringing their own usable oxygen supply on board during flight, arrangements must be made with commercial air carriers to provide in-flight oxygen. At least 48 hours' advance notice is usually required to arrange in-flight oxygen on U.S. domestic flights.

When arranging oxygen for patients who also use home oxygen, travel during business hours is advisable, in the event that personnel from oxygen vendors are needed to help with supplies during layover segments in the airport (where commercial air carriers do not supply oxygen). Flying nonstop is also preferable, to minimize both inconvenience and expense (especially if the air carrier charges for oxygen per flight coupon vs per oxygen bottle). Arranging a seat near the bathroom on the plane is advisable. If a nonstop flight cannot be arranged, pre- arranging for a wheelchair or motorized cart and oxygen (if needed on the ground) at the layover segment is advisable. Finally, several travel agencies specialize in arranging travel for patients with medical needs and can be useful contacts.[6,27-30]

Once again, because many details are necessary to arrange in-flight oxygen, it is advisable to reconfirm the arrangements at least 48 hours prior to the actual travel date.

Interacting with the Oxygen Vendors. Although in-flight oxygen is arranged with the air carrier, interacting with the traveler's personal oxygen vendor is also important to arrange oxygen on arrival and during layover segments. As noted earlier, commercial air carriers do not provide oxygen for travel other than actually aboard the aircraft. Circumstances under which oxygen would be required at the airport or on arrival at the destination include (1) when the traveler uses oxygen at home routinely, and/or (2) when the altitude of the destination is high enough that supplemental oxygen would be advisable even if oxygen has not been prescribed routinely at home.

Table 7. International Airline Carrier Requirements (as of December 1992) for Arranging In-Flight Oxygen*

Air Carrier	Advance Notice	Fee Structure†	Oxygen Supply		Traveler's Own O_2	Special Terms
			Liter Flow	Delivery Device		
Aer Lingus	24 h	None	2 or 4 L/min	Mask or cannula	Check empty as baggage	—
AirCanada	72 h	$59/Coupon	2 or 4 L/min	Cannula	Check empty as baggage	Carrier will call MD also
Air France‡	7 d	1,000 francs (about $173/ bottle)	2 or 4 L/min	Mask only	Check empty as baggage	Maximum of 2 3,250 L cylinders/ flight
Air New Zealand	72 h	$50/Coupon	2 or 7 L/min	NA	May bring one small canister for boarding/ deplaning	
Air Portugal	6 h	None	2 or 4 L/min	Mask only	Cannot transport own oxygen	One passenger with oxygen/flight
Austrian	NA§	None	2 L/min	Mask only	—	2 passengers with oxygen/flight limit
British Airways	72 h	30 lbs/journey	2 or 4 L/min	Mask only	Check empty as baggage	120- or 530-L cylinders
Canadian	5 days	$50 (U.S.)/ journey	2 or 4 L/min	Cannula only	May check empty as baggage but bring one D size (≤ 22-in length) cylinder on board	—
Finnair	72 h	None	2 or 4 L/min	Mask only	Cannot transport own oxygen	—
KLM	48 h	Must purchase extra seat but no charge for oxygen	0-6 L/min	Mask or cannula	May check empty as baggage	Carrier will call MD also. May charge extra seat for 3000-L cylinder
Lufthansa	24 h	NA	0-4 L/min	Mask or cannula	Cannot transport	Uses Dräger Pneumox concentrator
Mexicana	24 h	None	Passengers may use own O_2 supply			An attendant must accompany traveler
Sabena	48 h	None	Adjustable	Mask only	Check empty as baggage	—
SAS	1-6 h	None	2-4 L/min (310 L)	Mask	—	Maximum 6,000-L demand
			0-15 L/min (3,000 L)	Mask or cannula	Check empty as baggage	
Swiss Air	72 h	None	2 or 4 L/min	Mask only	Check empty as baggage	—
UTA	48 h	None	2 or 4 L/min	Mask only	Check empty as baggage	Maximum of 3 passengers/flight Accepts patients on stretchers

*From References 28 and 29, with permission.
†Local currency.
‡On 4 L/min with 320-L cylinder.
§NA = data not available.

Using a service or an oxygen vendor that can arrange extensive nationwide coverage maximizes the chance that oxygen can be supplied at all necessary destinations. Specific inquiries directed to the oxygen vendor will indicate whether such arrangements off-site are possible. Also, in interacting with the oxygen vendor, it is best to learn the types of systems that will be supplied because different vendors in different cities may provide different systems.

Personal Planning for the Travel Day. After making the arrangements specified above, several guidelines are appropriate for the actual day of travel (Table 5). The traveler should plan to arrive at the airport at least 1 1/2 hours early, partly because oxygen must be paid for on the day of travel. The charge structure for in-flight oxygen varies among commercial air carriers and is either on a per flight coupon (ie, per air ticket) basis or on a per bottle basis. As summarized in Tables 6 and 7, the per coupon and per bottle charges currently range from $40 to $75. Travelers should pack medications in their carry-on luggage, so that medications are accessible during flight. Also, having multiple copies of prescriptions and of physicians' letters handy is advisable. It is recommended that travelers bring their own nasal cannula and an extra length of oxygen tubing, to assure mobility during the flight and the use of a comfortable device. Not all commercial air carriers provide nasal cannula for use during flight (Tables 6 and 7), so having a personal cannula and a suitable adapter can assure availability.

Patients who use home oxygen are advised to arrange for oxygen during layover segments and on arrival at the final destination. Although these arrangements are best made through an oxygen vendor, it is possible that oxygen cannot be supplied through a vendor or that such arrangements have been overlooked in planning a hectic travel itinerary. Under these circumstances, oxygen-dependent travelers should know that oxygen may be available to them at First Aid stations at the airport; however, an informal poll of First Aid stations suggested that only 35% of those polled could provide oxygen.[4,27] As with other travel arrangements for the technology-dependent traveler, calling in advance to assure availability of transportation to the First Aid station (if expected) is strongly advised.

Finally, in addition to these tips, several informative documents can help patients in planning travel with oxygen. These include the American Lung Association Update (January 15, 1993) and an accompanying document entitled "Airline Travel With Oxygen,"[30] a booklet entitled "Requirements for Traveling with Oxygen" published by the American Association for Respiratory Care,[28] and the EuroLung Assistance Directory (available from the European Respiratory Society).[29]

Traveling with Oxygen by Car, Bus, and Rail

Just as travel with oxygen during commercial air travel is possible, so too is travel with oxygen by car, bus, or rail.[27] The optimal type of oxygen supply during travel over ground by car, motor home, bus, or rail is dictated by the patient's oxygen requirements.

For patients requiring continuous oxygen when traveling by car, large-volume liquid reservoirs are available and can be secured in the back seat of the car. Examples of these reservoirs include the Companion 21A (Puritan-Bennett, Lenexa KS), which has a 21-liter capacity and weighs 92 pounds when full, and the Liberator 20 (Cryogenic Associates, Bloomington MN), which contains 20 liters of liquid oxygen and weighs 85 pounds when full. Use in concert with an oxygen-conserving delivery device (eg, Oxymizer mustache or pendant, Oxymatic, Pulsair, or transtracheal catheter) can extend the duration of the available supply, though the bumpiness and heat of car travel will promote faster evaporation than with liquid systems used in the home. Travel with a backup E cylinder (which can provide approximately 5.8 hours of oxygen at 2 L/min) is advised to avoid running out of oxygen in case of a delay, breakdown, or detour en route.

For patients requiring oxygen at night only, an oxygen concentrator (eg, DeVO$_2$ MC 29 DeVilbiss, Somerset PA, or Freedom O$_2$, Roman Labs) can be rented and carried in the car for transfer to the hotel (or use in the motor home) overnight.

General precautions regarding travel with oxygen in the car include those shown below.

- Keep the car window ajar during travel to avoid creating an oxygen-enriched environment.
- Do not smoke or use the cigarette lighter.

- Avoid exposing the oxygen tank to excessive heat (ie, avoid storage in the car trunk or in an enclosed, parked car where temperatures can exceed 200° F).
- Secure cylinders during travel.

Traveling with oxygen on commercial bus lines is also possible. For example, Greyhound allows passengers to carry portable oxygen tanks on the bus and to check empty canisters as baggage.

Finally, travel with oxygen on commercial railroads is also possible. For example, Amtrak allows no more than 2 oxygen containers, each of which must weigh ≤ 75 pounds. Concentrators can be used on the train but must operate with a 12-hour backup supply that is not powered by on-board electricity. A supply adequate for 25% more than the expected trip duration is advised, and oxygen-dependent travelers are restricted to areas of the train reserved for nonsmokers.

Key contacts for arranging oxygen use during bus or rail travel are summarized in several publications.[27,28,31]

Cruising with Oxygen

Since 1985 when oxygen-dependent individuals first undertook a cruise,[27] cruising with oxygen has become increasingly popular and possible. As with air travel with oxygen, no standard regulations regarding cruising with oxygen exist and policies vary among cruise lines, some of which disallow oxygen completely, some admit unrestricted use, and some apply restrictions (Table 8). In general, cruise lines on which oxygen is allowed require patients to provide their own oxygen supply, although some cruise lines help contact local suppliers en route. As with arranging travel with oxygen on commercial air carriers, advance notice and clarification of all details are greatly advised. Practical tips regarding cruising with oxygen are reviewed in Table 9 and include

Table 8. Regulations Regarding Cruising with Oxygen (December 1992)*

Cruise Line	Advance Notice	Fee	Provide Own O_2?	Special Considerations
American Hawaii	≥ 1 wk	None	Yes	Cruise line can help contact suppliers en route
Celebrity	4-6 wk	None	Yes	—
Costa Cruise	NS†	None	Yes	—
Dolphin	4-6 wk	None	Yes	—
Holland-American	≥ 4 wk	None	Yes	No restrictions; physician letter required
Norwegian	≥ 4 wk	None	Yes	Concentrator and/or up to 6 E cylinders, no liquid allowed
Princess "Love Boat"	4-6 wk	None	Yes	—
Regency	4-6 wk	None	Yes	—
Royal Caribbean	4-6 wk	None	Yes	—
Royal Cruise	4-6 wk	None	Yes	—
Royal Viking	4-6 wk	None	Yes	—

*From References 28, 29, 31, with permission.
†NS = not specified.

- Providing advance notice, at least 4 weeks before departure;
- Obtaining a letter from the traveler's physician, specifying underlying diagnosis, fitness to travel, medications, and specific oxygen prescription (eg, delivery device, required flow-rate, and duration);
- Arranging the traveler's own oxygen supply in general;
- Arranging travel assistance insurance with coverage for pre-existing conditions and assurance of coverage for such things as medical expenses, repatriation costs, and costs for urgent return; and
- Arranging a cabin in a nonsmoking area of the ship, preferably near an elevator.

Specific questions about governmental regulations on cruising with oxygen should refer to regulation DOT-E 9856 and be directed to the Department of Transportation, Exemptions and Approvals Division [(202) 366-4535] or to the United States Coast Guard [Hazardous Materials Branch (202) 267-1577].

Table 9. Practical Tips about Cruising with Oxygen

Contact cruise line 4-6 wks before voyage (attention to Department of Special Services, Customer Service, or Operations).
Obtain local physician letter (approval to travel, prescription).
Usually arrange own oxygen supply (subject to cruise line restrictions), though cruise line may assist with contacting local suppliers en route.
Arrange "travel assistance insurance" with coverage for pre-existing conditions.
Arrange non-smoking cabin, preferably near elevator.
Direct questions to Department of Transportation about regulation DOT-E9856 and/or to United States Coast Guard Hazardous Materials Branch.

Travel with Technology Other Than Oxygen

Besides oxygen, individuals with chest disease may require other types of technologic support, including nebulizers and mechanical ventilators. A variety of portable, battery-run nebulizers are currently available and facilitate travel.

Although recreational travel poses formidable difficulties to ventilator-dependent individuals, mo-

Table 10. Air Travel with a Portable Mechanical Ventilator: Current Air-Carrier Policies*

Air Carrier	Portable Ventilator Accepted?	Special Considerations
American	Yes	Battery must fit under seat.
Continental	Not yet, but expect soon	—
KLM	Yes	Some planes have electrical hook-up for AC, but a KLM cable is needed.
Midway	Yes	Must fit under seat and use non-spillable batteries (dry- or gel-cell).
Northwest	Yes	Must fit under seat (space under seat varies). Some planes have electrical hook-up (may need battery pack).
Pan Am	Will consider on individual basis	—
Quantas	Will consider on individual basis	—
SAS	Yes, but needs MEDIF form†	All planes have electrical hook-up (either 115 V/60 Hz or 220 V/50 Hz).
Southwest	Yes	No electrical hook-ups (will need battery).
United	Will consider on individual basis	Dry- or gel-cell batteries only.
TWA	Yes	Must fit under seat and must be dry- or gel-cell battery.

*Reprinted from Lifecare Alert, 1993. Based on poll of 17 carriers regarding PLV-100 or PLV-102.
†Internationally approved medical information form.

tivated and well-informed ventilator-dependent patients have traveled successfully, including internationally by air.[32,33] Unlike the issue of travel with oxygen, few scholarly studies are available and even trade literature (eg, vendor newsletters) is scant. Based on one vendor's report,[32] which is summarized in Table 10, at least some commercial air carriers formally permit travel with a portable mechanical ventilator, and other carriers have transported ventilator-dependent air travelers despite lack of a formal policy. Table 11 summarizes several practical tips that apply when planning air travel with a portable mechanical ventilator.

Table 11. Practical Tips about Air Travel with a Portable Mechanical Ventilator

Check with carrier early (policies vary).
Make sure the ventilator will fit under the seat (space varies by aircraft).
Request back seat in section to get space between bulkhead and seat.
Make sure battery is dry- or gel-cell type.
Bring a spare ventilator on board.
Bring physician's letter summarizing condition and support needed.
Have the name of the supervisor in charge on the travel day.
For international travel especially,
bring spare U.S. standard nuts and bolts and
bring a transformer with 220-240 V input.

In Summary

In summary, recreational travel for the technology-dependent individual is common and includes travel via ground, water, and air, with oxygen and with mechanical ventilators. However, successful travel requires substantial advance preparation that involves fact-finding and communication with physicians, carriers, and vendors of equipment and oxygen. An increasing number of resource documents, both in the trade and in the scholarly literature, are available for relevant information and assistance.

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Stoller Discussion

Fletcher: Is ignorance bliss in this situation? When you look at the number of medical complications per flight, it's very low. How can we improve upon 1 in 30,000 medical complications—and those not necessarily respiratory? So if you have all these COPD patients traveling without consulting their physicians, and their P_{aO_2}s go down to 40 or 38, but they don't have complications from it, are we getting excited about nothing?

Stoller: That's an excellent point and perhaps it was not emphasized enough. I'm not necessarily espousing the current thinking. I tried to show you the assumption upon which it's based, and I'm struck, as you are, by the rarity of clinically significant events. My point is that clinically significant events may be uncommon, but this whole topic is not well known by the pulmonary community. Would you agree with that?

Fletcher: Yes! In fact, I didn't realize this whole body of information existed until the last day of the ATS meeting in Miami. I just chose to go to a lecture session and learned that this was a topic of interest.

Stoller: Exactly.

Fletcher: As a pulmonologist I wasn't aware of Henry Gong's work.

Stoller: It's extraordinary to me how poorly understood the topic is. These published reviews are helpful to me because they're enlightening as to what's out there that I know nothing about.

Fletcher: One other quick question. You did not discuss the liter flow in patients who are already hypoxemic and on oxygen who are going to engage in air travel.

Stoller: There's very little to guide that, except to suggest that you may want to set up hypoxic inhalation. In setting up such a system, you could selectively bleed in (at a simulated 8,000 feet of altitude) different liter flows and measure saturation. Because there are no regression equations for this issue, the only options are to make measurements. One can only imagine what it would be during hypobaric exposure, which is not available in most usual clinical circumstances, or by hypoxic inhalation, which itself is not easy to come by.

Mathews: I think there may be a couple of educational issues to be touched on here. I remember two incidents in the last 3 years—one in the air and one in the airport—where the airline personnel didn't know how to use their own oxygen systems. It's very distressing. A passenger got real bad on an aircraft, and they didn't know how to use this stuff. I looked at it, and I had never seen anything like it. We couldn't figure out how to turn the thing on. It turned out that when you pushed the oxygen connector in, that activated the flow. It was a set flow—a major problem. The following year, in the same airport, a patient being wheeled down the concourse was blue and gasping for breath. They had this wonderful full oxygen system on, but it was not delivering any flow. They said, "Well, we've got the thing in his nose. He's fine."

Stoller: My limited personal experience with medical problems encountered during air travel is similar.

Mathews: In fact, I've asked Puritan-Bennett if I could get some specs on the airlines. You know they make a lot of the aircraft safety equipment and oxygen systems. To date, I

haven't gotten any response, but I wanted to use that in our training program.

Stoller: The problem extends even beyond that. When one actually gets into what's in the medical kit on a commercial airliner, what's available to you isn't of much help. On the one occasion that I had to use the stethoscope on a plane, it was a stethoscope I would have bought my 4-year-old. You know, one of those kits that toy companies make.

Mathews: The last time I flew, there was a guy using his MDI; the staff on the airplane got all 'bent out of shape' because he was spraying an aerosol canister into his mouth.

Stoller: I think your point's well taken, Paul.

Hardy: I want to say a few words about applications in pediatrics. Hypoxic stressors such as air travel or visits to altitude can be a problem for pediatric patients with bronchopulmonary dysplasia (BPD).[1] Special concerns for pediatricians include length of flight, destination, and advance planning. General holiday confusion complicates air travel arrangements because special trips with sick infants often coincide with national holidays. Airlines don't usually have low-flow meters; so, tiny babies who might need something less than a 2-liter flowrate don't have that option through most carriers and vendors. The patients can't fly standby, which is sometimes an issue for families. They also need their own pediatric or neonatal nasal cannulas. Equipment arrangements need to include airport transfers and sometimes airline transfers. Portable systems need advance planning. Many pediatric pulmonologists do hypoxic challenges on these babies, who in my experience can be relatively stable in either room air or low-flow oxygen at sea level but with the challenges of altitude do very poorly. Very quickly, they can become apneic and hypoxemic. We frequently hear of infants and children with severe chronic lung disease who visit Lake Tahoe from San Francisco and decompensate.

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Gilmartin: I see a couple of problems when we get patients at our hospital in Denver from lower altitudes. Patients have been told by their physicians not to worry about the need for oxygen because the air is going to be thinner and they'll be fine. We see that quite a bit, and they're not all general practitioners that tell them this. So that's an educational issue. The other problem is that many of the patients don't have trouble on the flight with the cabin pressurized at 8,000 feet, but they get off the plane and then run into trouble. This may be related to the fact that on the plane they're not moving around at all, but when they land in Denver and start exercising they run into trouble. The patients end up many times in our triage because of that.

Stoller: I would agree with that, but I would say that the data that I've shown you in terms of the prevalence of medical events reflected both the in-flight and total airport experience. Now, they may have had difficulty as soon as they were in the cab leaving the airport. But the literature that exists, sparse though it may be,[1,2] does not reflect that scenario of someone having an acute problem on deplaning. These are rare events, and so finding differences among uncommon events may be statistically difficult.

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Sortor-Leger: In regard to that, I'm not sure how many people really understand the events. In discussing with some patients, it becomes obvious that they don't always associate their post-flight illness or difficulties with the flight itself until somebody suggests the connection for them.

Stoller: No question that there may be underreporting of those data. I think that one of the caveats in response to Fletcher's (Rick) comments is that one of the shortcomings of the epidemiologic data is that there may be significant underreporting of the things you might want to know about.

Sortor-Leger: Right, and in that case I'm not sure ignorance *is* bliss.

Stoller: Hard to know. It's certainly an area of controversy.

Dunne: I think the infrequency of catastrophic events in flight for people with chronic lung disease might be skewed by the fact that the current system often serves as a disincentive for a lot of people who probably would like to travel but choose not to do so. There's a big population out there that essentially never entertains the idea that they can travel. If that population gets out and starts traveling, you may see a different set of data. This may be only an observation as a result of what you so nicely illuminated, but there do exist certain obstacles when it comes to the availability of oxygen for travelling patients who really need it.

Make: Jamie (Stoller), I'm not sure you addressed the amount of oxygen needed during air travel in patients who are already receiving oxygen at sea level.

Stoller: There's no literature about it. I mean I'm unable to find anything that addresses that specific subset of patients.

Make: The ATS has attempted to develop a position statement on the need for oxygen during air travel but has not done so because of the controversies you reviewed. The Federal Register recently had a request for comments from the public based upon a couple of complaints from patients about the difficulty of receiving oxygen during air travel. The National Association of Medical Directors of Respiratory Care (NAMDRC) has enlisted some of the professional organizations (including the AARC, ATS, and ACCP) to comment in an attempt to solve the problems you talked about and to try to make it easier for people who do need oxygen when travelling.

AARC

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Novel Strategies for Delivering Oxygen: Reservoir Cannula, Demand Flow, and Transtracheal Oxygen Administration

Leslie A Hoffman RN PhD

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INTRODUCTION

Today, as many as 800,000 patients are receiving home oxygen therapy in the United States.[1] Most of these patients receive oxygen via a standard nasal cannula. Although simple to use, this device has a number of disadvantages. Oxygen is delivered during inhalation and exhalation, resulting in substantial waste.[2] The flow of oxygen can irritate sensitive nasal mucosa, and pressure from the tubing may cause ear and cheek tenderness or excoriation.[3] At flows greater than 6 L/min, the nasopharynx quickly fills with oxygen and higher flows cause minimal increase in the amount of oxygen delivered to the lungs.[4] Finally, the cannula is highly visible, and patients who are sensitive about their appearance may limit their use of oxygen in public and therefore reduce the benefits of pulmonary rehabilitation.

A number of oxygen-conserving systems have been developed in an attempt to resolve these problems. This paper reviews the mechanisms and devices available for use, efficacy, and costs of oxygen-conserving systems, including reservoir cannulas and demand-flow and transtracheal devices.

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ALTERNATIVES FOR LOW-FLOW OXYGEN ADMINISTRATION

Oxygen-Conserving Cannulas

The nasal cannula is a low-flow oxygen administration device. A patient breath is composed of 100% oxygen delivered via the cannula and a much larger volume of gas inhaled from the atmosphere. For a typical patient, a flow of 100% oxygen at 1 L/min adds approximately 3 to 4% to the inspired

oxygen fraction (F_{IO_2}). Thus, oxygen by nasal cannula delivered at 1 L/min increases the F_{IO_2} to about 24%, 2 L/min to approximately 28%, 3 L/min to about 32%, and 4 L/min to about 36%, depending on the patient's respiratory rate and tidal volume.[5] Although small, this increase in F_{IO_2} is sufficient to maintain the arterial oxygen saturation (S_{aO_2}) above 90% in the majority of patients with chronic lung disease.[5-7]

Mechanisms and Devices

Oxygen-conserving devices are capable of substantially decreasing oxygen requirement because they deliver a relatively pure bolus of oxygen early in the inspiratory phase of breathing. During a normal ventilatory cycle, approximately equal volumes of air are inhaled and exhaled but flow patterns are different. Exhalation occurs over a longer period of time than inhalation, resulting in a normal inspiratory-to-expiratory-time ratio (I:E) of 1:2. In patients with COPD, exhalation is prolonged and the I-E ratio can be 1:4, 1:5, or longer. The use of nasal cannulas wastes oxygen because all the gas delivered during the expiratory phase of breathing is lost to the atmosphere.

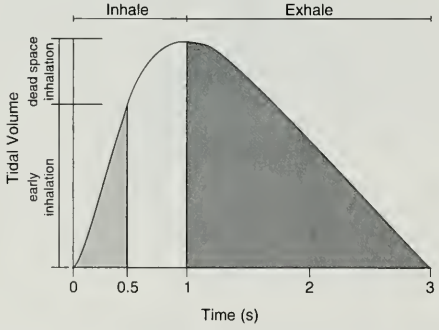

Fig. 1. Diagram of a tidal breath. A typical patient with COPD exhales for 2 seconds and inhales for 1 second. During the first half second of inspiration, the alveoli fill with air, much of which is gas that is rich in CO_2 (dead-space gas). The remaining fraction of the alveolar fill-volume is fresh gas from the room or an oxygen-delivery device. During the remaining half second of inspiration, the anatomic dead space fills with this fresh gas. Therefore, with the onset of exhalation, fresh gas is exhaled first and at end-exhalation alveolar gas (rich in CO_2) occupies the anatomic dead space. (Adapted, with permission, from Reference 8.)

In addition, of the gas that is inhaled, not all reaches the alveoli. As long as a patient breathes 20 times per minute and with no end-expiratory pause, each breath lasts 3 seconds. Of this 3 seconds, approximately 2 seconds is devoted to exhalation, 0.5 seconds to alveolar inhalation, and 0.5 seconds to dead-space inhalation (Fig. 1). The first 0.5 seconds of inhalation delivers the gas that is most likely to reach the alveolar level.[5,9]

The efficiency of oxygen administration can be increased by storing oxygen during exhalation for delivery during the early part of the next inspiratory phase or by timing delivery to occur during the early part of inhalation. Several types of oxygen-conserving systems attempt to accomplish this goal.

Reservoir Cannulas. Two types of reservoir cannulas are available—the oxygen-conserving nasal cannula (Oxymizer) and the oxygen-conserving pendant (Oxymizer Pendant, both from Chad Therapeutics Inc, Chatsworth CA). During inspiration, the patient who uses a reservoir cannula inhales a bolus of nearly pure oxygen at the beginning of inspiration, rather than the more dilute mixture (ie, 24-40%) delivered by a standard nasal cannula. Therefore, a lower flow of oxygen may be required to achieve the same S_{aO_2}. For example, a patient who uses oxygen at 2 L/min for an S_{aO_2} of 92% may only require 0.5 L/min when using the reservoir cannula. This decrease in flow results in a 75% savings in oxygen use.[9,10]

Oxygen-Conserving Nasal Cannula. The oxygen-conserving nasal cannula consists of a reservoir that is situated just below the nose, nasal prongs, and oxygen-supply tubing (Fig. 2). During exhalation, the reservoir expands and fills with dead-space gas. Oxygen from the delivery system then pushes the dead-space gas from the reservoir, resulting in a mixture of gas in the reservoir that is nearly pure oxygen. Early in inhalation, the reservoir collapses, delivering a high-F_{IO_2} bolus of gas.[9,10]

Oxygen-Conserving Pendant. The oxygen-conserving pendant consists of nasal prongs, wide diameter tubing, a reservoir that is situated on the patient's chest, and oxygen-supply tubing (Fig. 3). Operation is similar to that of the oxygen-conserving nasal cannula. During exhalation, dead-space

Fig. 2. Patient wearing an oxygen-conserving nasal cannula. (Reprinted, with permission, from Reference 8.)

gas fills the tubing and reservoir. At the same time, oxygen from the delivery system flows upward, pushing dead-space gas from the reservoir and tubing. During inhalation, the patient receives a bolus of nearly pure oxygen from the tubing.[9,11] Reservoir nasal cannulas are basically continuous-flow nasal cannulas that add a bolus of relatively pure oxygen just at the beginning of inspiration.[10]

Fig. 3. Patient wearing an oxygen-conserving pendant. (Reprinted, with permission, from Reference 8.)

Efficacy

A number of studies have evaluated the efficacy of reservoir cannulas (Table 1).[11-19] When comparisons were made between the use of a standard nasal cannula and reservoir cannula, results consistently showed that patients with obstructive lung disease achieved significantly higher mean S_{aO_2} when using a reservoir cannula at the same flowrate.[11-18] Mean S_{aO_2} also increased in patients with restrictive disease. However, the change was slightly less for these patients, compared to those with obstructive lung disease, and was not statistically significant.[15,17] Oxygen conservation results because the patient can be adequately oxygenated using lower flows. When flowrates required to maintain a similar S_{aO_2} were compared during use of a standard nasal cannula and a reservoir cannula, oxygen savings, in short-term studies under controlled conditions, ranged from 75% at lower flows to 50% at higher flows.[12-15] During exercise, savings were approximately 66%.[11,15] According to the manufacturer, the device should be replaced approximately every 3 weeks, which offsets oxygen cost-savings.

Although flowrate reduction is fairly predictable, factors such as nasal anatomy and breathing pattern can alter oxygen savings in individual patients. At low flows, both devices require the patient to exhale nasally to reset (open) the reservoir membrane. At higher flows, the oxygen flow into the reservoir is sufficient to reset the membrane.[10] In addition, reservoir cannulas may not function well in patients who use pursed-lips breathing (common practice during exercise).[10,18] Therefore, oxygen prescriptions should be determined by monitoring S_{aO_2} during rest and exercise.[11]

There has been limited evaluation of reservoir cannulas during long-term use. Claiborne and colleagues[16] compared oxygen use during 1 month of standard nasal cannula oxygen administration with 1 month of the pendant. With the pendant, oxygen use decreased 33%. After the costs of the device were deducted, total savings were $71/person/month. However, 9 of 21 subjects reported that they did not adhere to prescribed flowrates when using the pendant. In the 12 patients who adhered to the

Table 1. Selected Studies Comparing Oxygen-Conserving Nasal Cannula (CNC), Oxygen-Conserving Pendant (CP), and Standard Nasal Cannula (SNC) Delivery

First Author	Subjects	Outcome	Comments
Tiep et al,[12] 1984	Stable COPD, n = 20	CNC produced higher S_{aO_2} at rest than SNC ($p < 0.001$)	Short-term study, evaluation at rest
Tiep et al,[13] 1985	Stable COPD, n = 4	CNC produced higher S_{aO_2} ($p < 0.001$) at rest than SNC	Short-term study, evaluation at rest
Tiep et al,[14] 1985	Chronic lung disease, n = 7	S_{aO_2} was equivalent when using CP at 0.5 L/min and SNC at 1.8 L/min	Short-term study, evaluation at rest
Soffer et al,[15] 1985	Obstructive & restrictive, n = 20	S_{aO_2} during treadmill exercise using CNC comparable to SNC at about half the O_2 flow; less O_2 saved in patients with restrictive disease	Exercise evaluation, only 7 subjects with COPD completed exercise protocol
Carter et al,[11] 1986	Stable COPD, n = 10	With CP, S_{aO_2} during treadmill exercise was comparable to SNC at about 1/3 the O_2 flow	Short-term study using treadmill exercise
Claiborne et al,[16] 1987	Obstructive & restrictive disease, n = 21	CP acceptance was good in 12 and poor in 9 subjects; costs of O_2 decreased $141/subject/mo in 12 compliant subjects	Only long-term evaluation of CNC or CP; suggests lab O_2 savings > than home savings
Arlati et al,[17] 1988	Obstructive & restrictive disease, n = 16	With CNC, O_2 requirement decreased in subjects with obstructive disease; less O_2 saved in patients with restrictive disease	Short-term study using treadmill exercise
Tiep et al,[18] 1989	Stable COPD, n = 9	Specially designed pendant maintained S_{aO_2} during pursed-lips breathing	Pendant was modified so nasal exhalation was not required

protocol, total savings, after deducting equipment costs, were $141/person/month. These findings suggest that in some patients, potential cost-savings must be balanced against difficulties with acceptance and adherence.[16]

Patient Acceptance and Comfort

Because the quantity of oxygen needed when reservoir cannulas are used is less, the patient can use a portable oxygen supply that is smaller and lighter or a standard supply system for a longer time. However, many patients are concerned about their appearance when using oxygen and may find reservoir cannulas unacceptable.[20] For those less concerned about appearance, reservoir cannulas offer a number of advantages. The oxygen needs of patients with severe hypoxemia may be met using lower flowrates, resulting in less discomfort from high nasal flows[19] and a reduction in oxygen-supply requirement—the patient may need only 1 cylinder instead of 2 for a particular outing. Humidification is not necessary because of the reduced risk of mucosal drying when low flows are used. In fact, excessive moisture from humidifiers can impede the membrane action of the device and should not be used. The oxygen-conserving nasal cannula is softer and distributes the weight of the cannula over a

larger area of the face and, therefore, may allow healing of chafed or abraded skin on the face and ears.[10] Although less conspicuous, the pendant is heavier and more bulky and may increase facial and ear irritation.[16] Either device can be used short term during an acute illness to maintain S_{aO_2} until the underlying problem is resolved.

In Summary

- Reservoir cannulas are relatively inexpensive oxygen-conserving devices that are reliable and simple to use.
- At rest, oxygen savings in short-term studies under controlled conditions averaged 75% at the lowest flows and 50% at higher flows. During exercise, oxygen savings were approximately 66%.
- Oxygen flow can be reduced in patients with obstructive and restrictive disease, but the latter experience a smaller reduction in oxygen-flow requirement. If oxygen-flow requirement is reduced, even patients with severe hypoxemia may be adequately oxygenated using lower flowrates.
- Cosmesis and comfort may alter acceptance and, therefore, use of a device. The oxygen-conserving nasal cannula is comfortable but highly visible. The pendant is less visible but may cause ear and facial discomfort.
- Individualized oxygen prescriptions for rest and exercise are necessary because factors such as nasal anatomy, mouth breathing, and pursed-lips breathing alter efficiency of these devices.
- Little information is available in the literature regarding long-term performance or cost-savings of these devices.

Demand-Flow Oxygen Administration

Demand-flow oxygen delivery devices are designed to sense early inhalation or the crossover between exhalation and inhalation. A solenoid valve is triggered causing a pulse of oxygen to be delivered at this time. Precise timing allows the oxygen pulse to be delivered when it is most likely to reach the alveoli and thereby maximally conserve oxygen.[9,21]

Mechanisms and Devices

Demand-flow oxygen delivery devices can be pressure driven, flow driven, pressure and flow driven, or configured to alter the oxygen pulse in other ways.[21]

Companion Oxygen Saver (Puritan-Bennett Inc, Lenexa KS) delivers a metered dose of oxygen when inspiratory effort is sensed. The pulse ends within the first 25% of inspiration. Pulse volume is varied automatically based on the respiratory rate and oxygen flow setting. By varying the pulse volume based on respiratory rate and set flowrate, the system maintains a constant supplemental minute volume of oxygen over a range of 8-50 breaths per minute. Alarms indicate low battery and failure to sense inspiration. The unit automatically switches to continuous flow when a power failure or unit malfunction occurs. These devices are available for portable and stationary liquid systems, high pressure cylinders, and hospital wall outlets.[21,22]

Conservator (Penox Technologies Inc, Pittston PA) shuts off flow at the end of inspiration and activates flow at the start of the inspiratory cycle. Alarms indicate low battery, system failure, or failure to sense inspiration. The unit automatically switches to continuous flow when a failure occurs. Models are available for portable and stationary liquid systems and high pressure cylinders.[21]

Pulsair (Pulsair Inc, Ft Pierce FL) delivers a fixed volume per time per breath according to the setting selected. The amount of oxygen delivered increases as respiratory rate increases because each inspiration triggers delivery of the set volume. Alarms indicate low battery, low pressure, or failure to sense inspiration. One module is built into a portable liquid carrier that can be refilled from several manufacturers' reservoirs. A switch toggles to pulse or continuous flow, but there is no automatic switching in the event of failure. These are also available for stationary liquid systems, high pressure cylinders, and hospital wall outlets.[21,22]

Oxymatic (Chad Therapeutics Inc, Chatsworth CA) senses the beginning of inspiration and delivers a burst of oxygen over a short time. The pulse is

timed to interrupt flow during late (dead space) inspiration. The unit can be set to deliver an oxygen pulse during 1 of 4 breaths, 2 of 4 breaths, 3 of 4 breaths, or almost every breath. The system increases the flow of oxygen when respiratory rate increases because each inspiration triggers delivery of a given volume. The unit can be attached to a liquid system or high-pressure cylinder. Alarms indicate low battery and failure to sense inspiration. There is no mechanism for switching to continuous flow in the event of a system failure.[21,22]

Aluminum and Fiberwrapped Cylinders. Some companies market lightweight cylinders for use in combination with a demand-flow oxygen delivery system. *Oxylite* (Chad Therapeutics Inc, Chatsworth CA) consists of an Oxymatic and an aluminum or fiberwrapped cylinder. The Oxylite Mini weighs 4.5 lb and supplies oxygen for 10.5 hours at a setting equivalent to 2 L/min. Larger cylinders (7.5 lb) with a range of 28 hours at 2 L/min are also available.[20] *Pulsair Walkabout* (Pulsair Inc, Ft Pierce FL) consists of a Pulsair and an aluminum cylinder. The Walkabout Mini weighs 5 lb and supplies oxygen for 4 hours at a setting equivalent to 2 L/min. The Walkabout 2 weighs 8.3 lb and supplies oxygen for 10.5 hours at a setting equivalent to 2 L/min. Other sizes are also available.

Pulsing devices add a small additional weight to portable systems, but this is offset by the reduced oxygen requirement. Humidification is not necessary due to the low flows in use.

Efficacy

A number of studies have evaluated the efficacy of demand-flow oxygen delivery systems (Table 2).[22-31] When continuous-flow and demand-flow systems were compared, demand-flow oxygen delivery consistently resulted in a decrease in mean oxygen requirement during rest and exercise.[22-25,27-30] Oxygen savings, in short-term studies under controlled conditions, varied from 50-86%.[22-31]

Although demand-flow oxygen delivery has been shown to provide adequate oxygenation in most patients during exercise, some patients desaturate,[22,25,26] suggesting that device performance varies in individual patients. In a study to evaluate this potential, Braun and colleagues[22] compared S_{aO_2} during rest and exercise while subjects with COPD (n = 10) used a standard nasal cannula and 5 different demand-flow devices. Although device performance was comparable at rest, performance was highly variable during exercise. Three devices (Oxymatic, Pulsair, Companion Oxygen Saver) functioned best. However, in at least one subject, each device allowed desaturation to less than 80% during a 12-min walk. Also, each of the 6 delivery systems performed best in one subject. The authors attributed these results to differences in device function, mouth breathing, respiratory rate, and underlying lung pathology.

These findings, and those of other studies, reaffirm that an appropriate oxygen prescription cannot be guaranteed unless patients are tested during rest and exercise using the same delivery system they will be using at home.[22,25,26] This step is especially important in patients with restrictive disease because they tend to have rapid respiratory rates and short inspiratory-to-expiratory-time ratios (eg, 1:1). These breathing patterns result in complex pressure waveforms that can alter device function.[21]

One study evaluated efficacy of demand flow and continuous flow in patients (n = 100) hospitalized for diseases requiring oxygen therapy.[31] In an unblinded crossover design, pulsed and continuous oxygen were administered alternately during four 5.5-hour periods while S_{aO_2} was continuously monitored. Mean (SD) S_{aO_2} on demand flow was the same as for continuous flow—95.6 (2.7) % vs 95.3 (2.6) %. Because humidification was not necessary, costs associated with equipment ($23,936), personnel required to replace and refill the humidifiers ($19,459), and oxygen ($11,167) resulted in a net savings of $54,562. Most of the reduction in expense resulted from not using humidifiers, a practice that now applies to both continuous- and demand-flow systems during low-flow (< 4 L/min) oxygen administration.

Patient Acceptance and Comfort

Few studies have evaluated efficacy of demand-flow oxygen delivery during long-term use. Tremper and colleagues[27] followed 8 subjects with COPD during 3 months of demand-flow oxygen therapy. Mean oxygen consumption was reduced by 17% (range 9-28%). Six subjects had no complaints

Table 2. Selected Studies Comparing Demand-Flow Devices (DD) and Standard Nasal Cannula (SNC) Oxygen Delivery

First Author	Subjects	Outcome	Comments
Braun et al,[22] 1992	Stable COPD, n = 10	All devices functioned well for patients at rest, some (Oxymatic, Pulsair, COS-5) functioned better during exercise; each device allowed S_{aO_2} to fall to < 80% during a 12-min walk	Emphasizes need for individualized testing to determine rest and exercise flowrates
Mecikalski & Shigeoka,[23] 1984	Stable COPD, n = 10	DD reduced O_2 use (mean 28%, range 14-49%) compared to SNC	Short-term study on patients while at rest
Tiep et al,[24] 1985	Stable COPD, n = 12	Mean O_2 savings were 7:1, favoring the Oxymatic over SNC	Short-term study on patients while at rest
McDonnell et al,[25] 1986	Obstructive & restrictive disease, n = 16	During exercise, similar mean P_{aO_2} with less flow using Pulsair; at higher flowrates, some subjects had lower P_{aO_2} on DD	Short-term study during exercise, patient response was variable
Tiep et al,[26] 1987	Stable COPD, n = 9	During exercise, mean O_2 savings with Oxymatic were 87%; S_{aO_2} decreased 82% and 85%, respectively, in 2 subjects	Short-term study during exercise, patient response was variable
Tremper et al,[27] 1987	Stable COPD, n = 8	Mean O_2 use decreased 17% with Oxymatic, acceptance good in 6 of 8 subjects; no equipment problems in 3-mo trial	This long-term (3 mo) DD evaluation suggests that O_2 savings during lab are more than at home
Bower et al,[28] 1988	Obstructive & restrictive disease, n = 6	Compared to SNC, COS-5 Saver used 45% as much O_2 for rest, 44% for exerercise and 39% as much for sleep	RR decreased to < 8 during sleep in 3 patients & device switched to continuous flow
Senn et al,[29] 1989	Obstructive & restrictive disease, n = 18	Compared to SNC, COS-5 maintained similar P_{aO_2} at lower flow; during exercise, P_{aO_2} was higher on SNC in 1 subject and higher on DD in 1 subject	Short-term study during exercise, variable response
Carter et al,[30] 1989	Restrictive disease, n = 8	Higher mean S_{aO_2} during rest and exercise with Oxymatic	Short-term study during exercise
Kerby et al,[31] 1990	100 patients on O_2 in hospital	Mean S_{aO_2} on Companion 6 was the same as on SNC; potential cost-savings were $50,000/year	Of total, $11,167 was O_2 savings and remainder from not using humidifiers

and requested to continue using the device after study completion. Two subjects discontinued use due to noise of the device. In this study, oxygen conservation was substantially less than would be expected based on studies in which subjects were evaluated at one point in time in a laboratory setting. Because respiratory rate commonly increases during exercise, oxygen use (and therefore cost) increases, offsetting the cost-savings incurred during demand-flow oxygen delivery at rest. Subjects may also elect to continue using continuous flow, despite the availability of the demand-flow option.

In Summary

- Demand-flow oxygen delivery devices incorporate sophisticated electronics that can time the delivery of a pulse of oxygen early in the respiratory cycle.
- At rest, oxygen savings, in short-term studies under controlled conditions, averaged 50% to 86%. Oxygen savings were also experienced by patients during exercise, but response was more variable and some patients experienced severe desaturation.
- Individualized oxygen prescriptions for rest and exercise are necessary. In a study evaluating standard nasal cannulas and demand-flow delivery devices, each method performed best in at least one subject; however, each also allowed desaturation to < 80% in one subject.
- The combination of a demand-flow delivery device and lightweight aluminum or fiber-wrapped cylinder allows substantial mobility while using a device of minimal size and weight.
- Demand-flow devices make noise when pulsing and create a different flow sensation than do those used for continuous-flow oxygen delivery, factors that may decrease acceptance in some patients.
- Little information is available in the literature regarding long-term performance or cost-savings associated with these devices.

Transtracheal Oxygen Delivery

Mechanisms and Devices

Transtracheal oxygen administration, first introduced in 1982 by Heimlich,[2] delivers gas directly into the trachea via a small percutaneous catheter held in place with a bead chain necklace. Two types of transtracheal catheters are available: the Scoop (Transtracheal Systems, Englewood CO) and the Heimlich MicroTrach (Inmedco, Salt Lake City UT). With Scoop, transtracheal oxygen therapy is initiated in four phases—(1) patient evaluation, device selection, and teaching; (2) catheter insertion and stent; (3) in-place catheter cleaning (immature tract); and (4) catheter removed for cleaning (mature tract). With the MicroTrach, a stent is not used and the catheter is not removed for cleaning.

Several mechanisms are believed to alter oxygen requirement with transtracheal oxygen delivery. During inspiration, oxygen enters the trachea directly, reducing waste. During exhalation, oxygen fills the anatomic reservoir of the upper trachea and nasopharynx, storing a relatively pure bolus of oxygen for delivery during the initial phase of the next inspiration.[2,3] Transtracheal oxygen delivery also changes breathing pattern and ventilatory requirement. Most of the gas that reaches the lower respiratory tract is supplied by the catheter. This allows the patient to reduce inspired minute ventilation and inspiratory time.[32-36] During exhalation, the CO_2 concentration in the anatomic dead space is diluted by the continuous transtracheal flow.[35] Finally, the respiratory pattern of the diaphragm shifts to a less demanding pattern that may reduce the oxygen cost of breathing.[36] Similar changes are seen during insufflation of air and oxygen, suggesting that these outcomes result from the flow of gas directly into the trachea, rather than the type of gas used.[35,36]

Efficacy

A substantial number of studies have examined the use of transtracheal oxygen systems (Table 3).[2,3,32-52] Procedures for catheter insertion, patient management, and complications have been described.[2,3,38-44,46-49,51-62,64] In addition, a number studies have attempted to identify the effect of transtracheal oxygen delivery on breathing pattern and ventilatory requirement.[32-36] Reduction in oxygen requirement ranged from 37-58% at rest, and in one study was 30% during exercise.[38,42,46,51] Oxygen savings decrease with exercise because the size of the anatomic reservoir is fixed and there is no mechanism to store additional oxygen to meet exercise requirements.

In addition to conserving oxygen, transtracheal systems have several unique advantages and disadvantages when compared to other oxygen-conserving systems. Potential benefits are

- elimination of nasal drying, epistaxis, ear and facial tenderness, or excoriation;[61]

Table 3. Selected Studies Describing Outcomes during Transtracheal Oxygen Delivery (TTO)

First Author	Subjects	Study Design	Outcome
Christopher et al,[42] 1987	COPD, n = 77; other diagnoses, n = 23	Prospective with ≤ 2 yr follow-up	With TTO, mean O_2 requirement from 2.3L/min to 1 L/min, 30% less O_2 flow used with exercise; hematocrit decreasd significantly in 12 subjects
Bloom et al,[44] 1989	COPD, n = 43	Randomized to TTO or SNC,* with 6-mo follow-up	ADL† increased ($p < 0.01$) as evaluated by psychometric tests; TTO patients purchased 4.5 times more O_2 cylinders/month ($p < 0.0001$); hospital days decreased in TTO group and increased in SNC group
Bell et al,[45] 1988	COPD, n = 15	Treadmill exercise pre- and 6-mo post-TTO	Exercise capacity was unchanged with TTO
Hoffman et al,[48] 1992	COPD, n = 20	Prospective comparison of 6-mo using SNC and 6-mo using TTO	With TTO, O_2 requirement was 45% less at rest and 39% less than expected (mean 14%); hospital days decreased ($p < 0.002$) and exercise tolerance improved with TTO use ($p < 0.0001$)
Hoffman et al,[49] 1993	COPD, n = 32	Randomized to TTO, SNC, or OX‡ with 12-mo follow-up	12-min walk distance increased ($p < 0.04$) over time for TTO subjects compared to a reduction for patients using SNC and OX; TTO subjects experienced less dyspnea compared to OX and SNC subjects; hospital days tended to be less in TTO—mean (SD) 3.7 (9.1) compared to SNC 5.7 (10.6) and OX 8.1 (15.8), but no differences were statistically significant
Wesmiller et al,[50] 1990	COPD, n = 11	Randomized double-blind trial comparing 12-min walk distance with SNC & TTO	Subjects completed 12-min walks using O_2 via TTO (usual flow by TTO + 0.5 L/min by SNC) and SNC (usual flow by SNC + 0.25 L/min by TTO); two tanks were used to disguise delivery method; S_{aO_2} was not different but walked distance was greater in the TTO group ($p < 0.01$)
Adamo et al,[51] 1990	Obstructive & restrictive disease, n = 21	Prospective with 20-mo follow-up (162.5 patient-months)	Substantial number of complications: procedural-technical (n = 26), symptomatic (n = 51), infectious (n = 17), and miscellaneous (n = 24); three patients stopped TTO; adverse events common but serious events were uncommon (10/120)
Rai et al,[52] 1993	Obstructive & restrictive disease, n = 3	Second part of consecutive 51-patient sample	Fewer procedural complications increased experience using TTO; no change in number of symptomatic or infectious complications

*SNC = standard nasal cannula.
†ADL = activities of daily living.
‡OX = OxyFrames.

- improved adherence with continuous oxygen use;[42,44]
- increased exercise tolerance,[44,48,49]
- adequate oxygenation in refractory hypoxemia (Fig. 4);[43,47,51]
- reduced hospitalization;[44,48] and
- concealed oxygen delivery.

Fig. 4. Patient with a transtracheal oxygen catheter in place. (Reprinted from Reference 63.)

There are disadvantages associated with transtracheal oxygen delivery: high cost and increased risk of infection and complications.

- High cost—Transtracheal oxygen administration is unlikely to be successful unless patients are carefully selected, receive detailed teaching, and are monitored at frequent intervals during the initial weeks of use. This increases personnel costs. Reimbursement is available for the insertion procedure and connection to oxygen but not for replacement catheters. The manufacturer-recommended interval for catheter replacement is every 3 months.
- Increased risk of infection—While most series have reported a low incidence of infection,[39,42,47,48] others have described more frequent lower respiratory tract infections during transtracheal oxygen delivery, particularly in patients diagnosed with interstitial lung disease.[51,56]
- Increased risk for complications—Problems and complications are rare during placement and during the stent period and are relatively uncommon when the catheter is removed from a mature tract (about 8 weeks after insertion) for cleaning. Most complications occur when the tract is immature and the catheter is cleaned in place.[42,43,46-49,51-62] The most serious complication, mucus balls, results from accumulation of inspissated secretions on the outside of the catheter.[61] If not removed by cleaning, mucus balls increase in size. There have been several reports of patients who experienced severe respiratory distress or acute respiratory failure from mucus balls, including one fatality.[54,55,57-60] Patients should be taught to recognize the signs and symptoms of mucus-ball accumulation (ie, back pressure resulting in release of the humidifier's high-pressure-relief valve, severe cough, unexplained dyspnea, hemoptysis, and mist in the oxygen-supply tubing) and, after connection to oxygen, called at periodic intervals or asked to return to the clinic to assess catheter patency.[3,47,61,63] Mucus balls can be removed by stripping the catheter, a procedure that involves removing the catheter over a guide wire.[3,61,62] If the patient is at high risk for mucus-ball accumulation (high flow, weak cough) and cannot return for checkups, stripping can be done in the home via referral to a home care agency.[62]

Heimlich & colleagues[39] and Christopher & colleagues[42] reported fewer problems and complications from a 200- and a 100-patient series than have been reported in smaller series,[46,48,51] suggesting that developing expertise in the management of patients who use transtracheal oxygen involves a "learning curve." Rai and colleagues[52] compared complications in the first 21 and subsequent 30 patients in their series and found a decreased incidence of procedure-related complications but no change in the incidence of airway complications or infectious complications.

To some extent, problems and complications can be decreased by appropriate selection of candidates.

Table 4. Transtracheal Oxygen Delivery Evaluation in Refractory Hypoxemia*

To evaluate the potential for benefit with transtracheal oxygen delivery in patients with refractory hypoxemia ask:

Is the patient receiving optimal therapy? If not, can therapy be altered and oxygen requirement decreased?

What is the current oxygen requirement? Is it anticipated that oxygen requirement will remain relatively stable or will it escalate?

If the patient is in a preterminal phase, will transtracheal oxygen delivery be more likely to complement or complicate management?

What P_{aO_2} is achieved with administration of 100% oxygen? Does this P_{aO_2} suggest that the patient will benefit from transtracheal delivery? For example, consider two patients who remain hypoxic on maximal nasal oxygen delivery. While breathing 100% oxygen, Patient A's P_{aO_2} is 150 torr and Patient B's P_{aO_2} is 50 torr. Patient B is not likely to benefit from the transtracheal administration of oxygen because it would not increase the P_{aO_2} beyond that achieved with 100% oxygen via a nasal device.

Can the patient walk? The ability to walk typically equates with the ability to generate sufficient airflow to clear mucus balls by coughing.

Will the patient be able to return to the healthcare institution to perform periodic stripping of the catheter or is care available in the home?

*From References 3 and 62, with permission.

The best candidates are patients who require low-flow oxygen because they are more likely to benefit from improved exercise tolerance. While transtracheal oxygen administration can reverse refractory hypoxemia, all patients are not good candidates for this therapy (Table 4).[43,47,62]

When mechanical ventilation is required in a patient using transtracheal oxygen, intubation should be performed with the catheter in place. After intubation, the catheter should be capped, left in the stoma, and the site cleaned as often as required to maintain a clean appearance. Irrigation is not necessary. After extubation and before use, the catheter should be irrigated and stripped to remove any mucus accumulation.[37] Insufflation of gas through the catheter decreases inspired minute ventilation and may assist in weaning.

Patient Acceptance and Comfort

The majority of patients who use transtracheal oxygen delivery systems have been extremely enthusiastic about their use.[38-49,51] Patients report less shortness of breath during exertion and increased mobility. Elective catheter removal has been lower in larger series.[38,42] Follow-up in some series extended to ≥ 7 years.[39,47]

In Summary

- Transtracheal oxygen systems deliver oxygen directly into the trachea, resulting in a more efficient and less-visible method of oxygen delivery. At rest, oxygen savings averaged 37-58% and during exercise 30%. In addition to conserving oxygen, transtracheal oxygen delivery alters breathing pattern in ways that can reduce the oxygen cost of breathing.
- The use of transtracheal oxygen can eliminate discomfort from the nasal cannula, improve adherence to continuous oxygen use, increase exercise tolerance, and reduce hospitalizations.
- Patients with refractory hypoxemia may be adequately oxygenated without use of a face mask. Careful selection is necessary because not all patients are good candidates for this therapy.
- Disadvantages include the risk of complications, increased time needed for candidate evaluation and teaching, and cost of replacement catheters. Close patient follow-up is mandatory during the initial weeks of use.

Combined Demand-Flow and Transtracheal Oxygen Delivery

Mechanism and Devices

Demand-flow and transtracheal oxygen systems can be combined to further increase oxygen conservation. Several potential benefits could result from combining demand-flow and transtracheal oxygen administration. Pulses are muffled inside the trachea, thereby reducing the noise normally associated with demand-flow therapy. Also, the additional oxygen savings may further decrease oxygen re-

quirement, allowing the patient to use a smaller, lighter, portable device and, ultimately, perhaps a battery-powered concentrator.[8]

Efficacy

Only a few studies have evaluated the potential of pulsed transtracheal oxygen delivery. In a short-term study under controlled conditions, Tiep and colleagues[65] compared oxygen flows during pulsed and continuous transtracheal flow in 17 subjects. The subjects required less oxygen with pulsed flow (0.4 L/min) compared to continuous flow (1.2 L/min). Similar findings have been reported in preliminary reports by others.[66,67]

In a long-term comparison of these technologies, Yaeger and colleagues[68] evaluated response to pulsed and continuous transtracheal flow during 2 separate nights using standard nocturnal polysomnography and during 1 month of either continuous nasal cannula or pulsed nasal cannula oxygen administration, plus 1 month of continuous transtracheal oxygen administration and 1 month of pulsed transtracheal oxygen administration. Pulsed nasal cannula oxygen administration was evaluated in 6 subjects, continuous nasal cannula oxygen administration in 14 subjects, and pulsed and continuous transtracheal oxygen administration in 20 subjects. Six subjects were not adequately oxygenated on pulsed nasal oxygen administration during exercise. During long-term follow-up, all remaining subjects were adequately oxygenated during pulsed-delivery oxygen therapy as determined by S_{aO_2} and a stable hematocrit over the study period.

Patient Acceptance and Comfort

Subjective evaluations by patients who used pulsed transtracheal oxygen were highly variable. Some subjects claimed to be more energetic and some claimed that they were less energetic while using the various modes of oxygen delivery. Since S_{aO_2}, hematocrit, and disease state were stable during the study period, this finding is difficult to explain. All subjects had a strongly stated preference for transtracheal oxygen-delivery methods, a factor that may have influenced their evaluations of energy level when using oxygen by pulsed and continuous nasal delivery. Alternately, other unidentified factors may have been involved.

In Summary

- Pulsed transtracheal oxygen administration has the potential to further reduce oxygen requirement in comparison with pulsed nasal cannula flow.
- Few studies have evaluated outcomes with pulsed transtracheal oxygen administration, making it premature to evaluate benefits and risks of this approach.

COST ISSUES

A number of variables must be considered when evaluating savings from oxygen-conserving systems (Table 5). It is well established that oxygen administration by reservoir cannulas and demand-flow and transtracheal systems can decrease the amount of oxygen used by patients.[9-19,21-30,38-44,46-49] However, each of these oxygen-conserving systems also increases costs. The patient and/or home medical equipment supplier must provide equipment that is more expensive than the standard nasal cannula and continuous-flow delivery system. The equipment is also more complex and, therefore, requires more instruction and supervision.

Table 5. Comparison of Oxygen Conservation Methods in Relation to Selected Characteristics*

Characteristic	Reservoir Cannual	Electronic Demand Device	Transtracheal Catheters
Cosmetics-appearance	+	++	+++
Oxygen savings	++	+++	+
Device cost	+++	++	+
Comfort	++	++	+++
Device reliability	+++	+	++
Nose and ear irritation	++	++	+++
Complications	++	++	+
Noise	++	+	+++
Humidification	+++	++	++

*From Reference 20, with permission. + = least desirable; ++ = somewhat desirable; +++ = most desirable.

When evaluating costs of home oxygen therapy, it is important to remember that most of the expense of home oxygen therapy results from equipment and delivery costs. Therefore, a reduction in flowrate

has little effect on costs unless this reduction also decreases the frequency of delivery of liquid oxygen or the number of cylinders the patient uses for ambulatory oxygen therapy.

Other considerations relate to reimbursement. If reimbursement were directly related to the amount of oxygen used, reduction in use would translate to a lower monthly bill. However, home oxygen therapy is typically reimbursed on a fixed-fee basis. There is a fixed payment for the equipment and a fixed payment for oxygen based on delivery flow-rate where the base rate is the amount reimbursed for oxygen delivery at 1-4 L/min. At flows > 4 L/min, reimbursement is 1.5 times the base rate and the payment for oxygen at flows < 1 L/min are 0.5 the base rate.[1] This fixed-fee system creates a disincentive for development of oxygen-conservation systems. When conservation systems reduce oxygen requirement to < 1 L/min, reimbursement will be 50% less.

In long-term evaluations of oxygen-conserving systems, results have shown savings but less than expected. For example, Tremper and colleagues[27] reported a mean oxygen savings of 17% (range 9-28%) during use of a demand delivery device. Claiborne and colleagues[16] reported a 33% reduction in oxygen use in patients using an oxygen-conserving pendant, with a further decrease in fully compliant patients. My colleagues and I[48] reported a 14% reduction (range +4% to –32%) oxygen use during transtracheal oxygen administration compared with standard nasal cannula oxygen administration. Bloom and colleagues[44] reported that subjects randomized to transtracheal oxygen purchased 4.5 times more tanks of compressed gaseous oxygen per month than they purchased when using a nasal cannula. Thus, 'real-life' savings from oxygen-conservation systems have been less than suggested by short-term studies.

There are a number of explanations for these findings. Oxygen-conserving systems are believed to improve adherence with a 24-hour regimen and increase use of oxygen outside the home. Both outcomes increase oxygen use because oxygen is used for a longer duration and flows during exercise are typically higher than at rest. Improved adherence to 24-hour oxygen use and increased exercise can improve physical and psychological outcomes and thereby greatly improve the quality of life for patients with chronic lung disease. In turn, morbidity and mortality may decrease. Cost-benefit considerations need to focus on outcomes that are broader than change in oxygen use, including whether such devices alter quality of life, functional status, or use of healthcare resources.

SUMMARY

- Oxygen-conserving devices can decrease costs because less oxygen is used. However, they may also add costs due to the more complex equipment involved (ie, device and catheter replacement).
- Oxygen is relatively expensive. To decrease the cost of oxygen therapy, conserving devices must decrease the frequency of home deliveries and/or the number of cylinders used.
- By promoting mobility and improving adherence to continuous oxygen therapy, oxygen-conserving systems may increase the amount of oxygen used, thus offsetting savings.
- It is shortsighted to focus only on reducing the costs of oxygen administration. Attention also needs to be directed to patient outcomes such as functional status, use of healthcare services, and quality of life.

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See Page 386 for the Discussion that followed the presentation of this paper.

Timing of Tracheotomy: What Is the Best Approach?

Leslie A Hoffman RN PhD

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Introduction

The decision as to when, in the course of treatment, tracheotomy is indicated is based on the clinician's expectations regarding the patient's ability to ultimately recover and the length of time that this recovery will require. The timing of tracheotomy—early or late—may, of itself, have a major effect on the patient's recovery. In several studies of patients with multiple trauma, early tracheotomy was associated with a reduction in the duration of mechanical ventilation, intensive care unit/hospital stay, and incidence of pneumonia compared with prolonged endotracheal intubation.[1-3] However, tracheostomy may also predispose patients to increased morbidity and mortality for a variety of reasons (Table 1).[4] In addition, cost of care is increased by the required surgical procedure. The purpose of this paper is to review the available information regarding prolonged endotracheal intubation, the timing of tracheotomy, and decannulation/extubation.

Table 1. Major Complications of Tracheostomy*

Early Complications	Late Complications
Aerophagia	Aspiration
Accidental decannulation	Increased incidence of respiratory infection
Pneumomediastinum	Pneumonia
Subcutaneous emphysema	Stomal infection
Aspiration	Swallowing dysfunction
Incisional hemorrhage	Tracheal stenosis
Pneumothorax	Tracheal granuloma
	Tracheo-innominate fistula
	Tracheo-esophageal fistula
	Tube obstruction

*Adapted from Reference 4, with permission.

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Perspective on Prolonged Endotracheal Intubation

While there is ample reason for concern about the incidence of tracheal injury, complications can also arise from laryngeal injury. The two major mechanisms that are believed to cause laryngeal injury from an endotracheal tube (ETT) are tube movement causing abrasion of the mucosa and pressure necrosis.[5] Pressure necrosis generates ulcerative inflammation that may progress to chronic glottic incompetence or laryngeal stenosis, resulting in dysphagia or aspiration following extubation.[6] In some patients, laryngeal stenosis has been severe enough to require reconstructive surgery.[7] Other sequelae include persistent hoarseness and/or sore throat, hemoptysis, vocal-cord granulomas, and vocal-cord paralysis.[8,9]

When an ETT is inserted, it must bend to conform to the shape of the airway (Fig. 1).[10] An ETT primarily exerts pressure at three locations: the posteromedial portions of the true vocal cords, the posteromedial surface of the arytenoid cartilages, and the posterolateral aspect of the cricoid cartilage.[5,11-14] Potential for laryngeal injury is believed to be greater if there is excessive movement of the tube against the larynx, such as occurs during neck flexion and extension, coughing, swallowing, and attempted phonation.[14] Women have a smaller larynx than men and thus may be at greater risk for laryngeal injury.[14]

The loading force, a measure of the force exerted on laryngeal structures due to deformation of an ETT, varies with the shape and size of the ETT and materials used in tube manufacture.[13,14] Red-rubber (Rusch) tubes, commonly used in the 1960s, exerted a high tracheal loading force (1,000 g).[13,15-17] Early tracheotomy was used to minimize the potential for laryngeal injury from these tubes. In the 1970s, polyvinyl chloride and silastic tubes were introduced. These tubes exerted a much lower loading force (200 to 500 g), thereby making the option to delay tracheotomy in favor of prolonged endotracheal intubation more attractive.[13,15-18]

Laryngeal injury is primarily pressure-induced and is obviated by tracheotomy. After a tracheotomy is performed, pressure-induced injury resulting from cuff inflation can be minimized by frequent monitoring and adjustment of cuff pressure.[19]

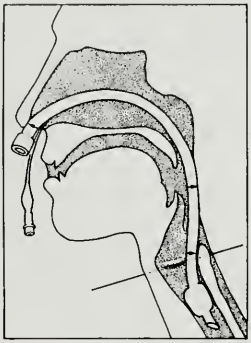

Fig. 1. Points of pressure-induced trauma with nasal and oral endotracheal tubes. A. Arrows indicate the direction of force with nasal endotracheal tubes. B. The arrows in the cross-sectional view taken at the level of the glottis (transverse line in Fig. 1A) indicate areas within the airway that are subject to pressure from both nasal and oral endotracheal tubes. (Reproduced, with permission, from Reference 10.)

Perspective on Early Tracheotomy

There is no consensus with regard to the optimum time to perform a tracheotomy. When making a decision about timing, the physician must weigh potential risks (Table 1) against potential benefits (Table 2). If the duration of endotracheal intubation is expected to be brief, tracheotomy is typically delayed. Conversely, early tracheotomy is usually performed if it is likely that the requirement for endotracheal intubation will persist for several weeks. Most patients fall into a 'gray' area somewhere between these points while the decision to perform tracheotomy is re-evaluated and often delayed, on a day-to-day basis, anticipating extubation 'tomorrow.'

Table 2. Potential Benefits of Tracheostomy

Benefit	Mechanism
Improved comfort	Airway fixation method is simpler and not applied to the face.
Improved mobility	Travel outside the ICU is possible using a manual resuscitation bag or portable ventilator. Bed-to-chair mobility is enhanced.
Improved secretion clearance	Secretions are more easily removed by suctioning and less strength is required to expectorate secretions from the airway than with an endotracheal tube.
Mouth care is facilitated	Because there is no endotracheal tube in the mouth, oral secretions are likely to decrease and there is no obstruction to mouth care.
Improved communication ability	Patient can use fenestrated tracheostomy tube, speaking valve or 'trach talk.' Lips can mouth words. Psychological benefits from improved communication may motivate patient recovery.
More secure airway	Lower incidence of accidental decannulation with tracheostomy compared to ETT.[8]
Lower incidence of pneumonia and less-serious respiratory infections	Reports in trauma patients[1-3] have shown a significant association between the reduced incidence of pneumonia and less severe respiratory tract infections and early tracheotomy.
Fewer days of mechanical ventilation	Reports in trauma patients[1-3] have shown a significant association between fewer days of mechanical ventilation and early tracheotomy.
Lower risk of laryngotracheal injury in high-risk patients	Factors that increase the risk of laryngotracheal pathology are believed to include head rigidity, frequent neck flexion and extension, attempted phonation, swallowing, and accidental extubations.[6]

Perioperative Complications

Tracheotomy is a surgical procedure that poses risks of potentially serious early and late complications. Intraoperative complications of a tracheotomy include hemorrhage, pneumothorax and pneumediastinum, recurrent laryngeal nerve injury, and cardiorespiratory arrest.[20] Complications that may occur in the immediate postoperative period include hemorrhage, stomal infection, subcutaneous emphysema, tube obstruction, and tube displacement.[20] Because these complications can be avoided if tracheotomy is not performed, estimates of how frequently they occur are an important consideration when evaluating the risks and benefits of early tracheotomy.[6,18]

The Literature. Most series that exclude emergency tracheotomy from analysis and confine procedures to a core team of experienced clinicians report low complication rates.[6,18] Stock and colleagues[21] prospectively reviewed the outcome of 81 critically ill patients who underwent tracheotomy. No major morbidity was reported. Minor complications included cardiovascular instability (1.2%), wound hemorrhage that was controlled by packing (2.4%), and subcutaneous emphysema (2.4%). Dane and King[22] prospectively evaluated 40 patients requiring mechanical ventilation who underwent tracheotomy and reported that 10% developed stomal or intrabronchial hemorrhage. Two (5%) of these patients had severe liver disease and abnormal coagulation mechanisms. All responded to bleeding-control measures applied locally. Similar rates of clinically important complications have been reported from other series in which a limited number of clinicians performed the procedure.[4,23-25] When management was not confined to a core team of clinicians, more complications were reported. In a prospective series of 150 patients, 51 of whom underwent tracheoto-

my, Stauffer and colleagues[8] reported an increased incidence of complications that included stomal hemorrhage (36%), stomal infection (36%), subcutaneous emphysema (9%), stomal erosion (9%), excessively long skin incision (8%), and pneumomediastinum (4%). Two patients (4%) experienced a cardiopulmonary arrest, one from tube misplacement at the time of tracheotomy and one 5 days post tracheotomy when the tube inadvertently dislodged. In this series, care was provided by teams of attending physicians, house staff, and medical students.

In Summary.

- Available data indicate that the incidence of early complications after tracheotomy varies among institutions.
- If the incidence of perioperative complications is low, concern about potential complications should not be a reason to defer tracheotomy in favor of prolonged endotracheal intubation.

Laryngeal Complications

Laryngeal complications can be avoided if early tracheotomy is performed. Therefore, estimates of how frequently these complications occur are an important consideration in evaluating the risks and benefits of early tracheotomy.

The Literature. Some degree of laryngeal damage is probably inevitable, because ETTs exert pressure against the mucosal surface as high as 400 mm Hg in animal models.[26] However, all patients do not experience significant sequelae.[6-9,14,18,27-30] Patients who are moving about, attempting to speak, and frequently swallowing or coughing are believed to be at greater risk, because these movements can increase mucosal abrasion from the ETT.[7,14] Another variable believed to increase risk is duration of intubation. If the amount of time an ETT was present in the airway was found to directly influence the degree of laryngeal injury, specific recommendations could be made regarding timing of tracheotomy.

Whited[7] examined 200 patients who were intubated for 2-5 days (n = 50), 6-10 days (n = 100), or 11-24 days (n = 50) at three intervals: extubation, weekly or biweekly until they were discharged from the hospital, and again 3-6 months after discharge. Patients who were intubated for 11-24 days had a 12% incidence of laryngeal stenosis, whereas the incidence was 5% in patients intubated 6-10 days and 2% in patients intubated less than 6 days. Consequently, Whited[7] recommended tracheotomy be performed after 10 days of ETT intubation.

Other studies have not found the same magnitude of correlation between duration of intubation and long-term laryngeal injury. Dunham and LaMonica[29] randomized 74 trauma patients to Day 3 versus Day 14 tracheotomy. No significant differences were found in the number of complications in the early or late group. Colice[30] evaluated the incidence of laryngeal injury in 54 patients who had required intubation for a mean (SD) of 9.3 (5.1) days. Each patient was examined by direct laryngoscopy within 24 hours of ETT removal (at extubation or after tracheotomy) and every 2 weeks thereafter until the examination was normal, the patient died, or abnormal laryngeal healing was recognized. Only 5 patients (9%) had a normal larynx at extubation. However, in the remaining patients, most lesions (78%) healed without surgical intervention. Three patients (6%) died before complete healing and 4 (7%) developed laryngeal granulomas that required surgical intervention in all but 1 case. The findings of this study suggest that laryngeal injury is common but not of serious, long-term consequence in the majority of patients. Kastanos and colleagues[9] also reported a high incidence (12/19) of laryngeal lesions (mainly true vocal-cord granulomas and vocal-cord ulcerations) in critically ill patients intubated for an average (SD) of 6.2 (3.7) days. Four months later, established laryngeal lesions were present in 3 patients. No relationship existed between laryngeal injury and age, sex, duration of intubation, and/or number of intubations. Similarly, Stauffer and colleagues[8] found that survivors of long-term intubation rarely experienced long-term laryngeal morbidity.

Individual differences probably account for some of the variability in occurrence of complications. In critically ill patients, factors such as hypoxemia, hemodynamic instability, and nutritional depletion alter tissue repair and oxygen delivery and, hence, potential for tissue damage. In neurological patients, the incidence of major largynotracheal pathology was higher in rigid, head-injured patients than in those who were not head-injured or were head-

injured but not rigid ($p < 0.001$).[29] Frequent neck flexion and extension, combativeness, seizures, and accidental extubations may also increase risk of injury.[14] The combination of prolonged intubation, followed by tracheotomy, may also increase risk. After tracheotomy is performed, the vocal cords cease to move, allowing the inflamed posterior larynx to scar and stricture in the absence of the stenting effect of an ETT and the force of laryngeal opening during respiration that acts to keep the cords apart.[5]

In Summary.

- Most patients experience some degree of laryngeal damage as a result of endotracheal intubation.
- Although common, laryngeal injury does not cause serious long-term morbidity in most patients.
- In most studies, no relationship was found between duration of intubation and incidence or severity of laryngeal complications.
- The potential for airway injury does not appear to be solely related to the duration of endotracheal intubation.

Nosocomial Infection

Both endotracheal intubation and tracheostomy increase the risk of aspiration and hence the potential for colonization of the airways and nosocomial infection. However, critically ill patients do not appear to be at increased risk of airway colonization and nosocomial pneumonia, independent of other variables.[6,18]

The Literature. El-Naggar and colleagues[31] randomized 52 patients with acute respiratory failure to Day-3 tracheotomy versus continued endotracheal intubation and found similar colonization during the first 4 days of intubation. Subsequently, patients who were converted to tracheostomy had a larger number of new pathogens cultured from their tracheal secretions compared to patients continued on endotracheal intubation.

However, Dunham and LaMonica[29] reported no difference in the incidence of tracheitis, pneumonia, or peristomal infection in patients randomized to tracheotomy on Day 3 versus Day 14. Also, Oswood and colleagues[32] reported no difference in the results of quantitative cultures of laryngeal and tracheal secretions obtained within 12 hours before and 5 days after tracheotomy.

In addition, three studies in patients with traumatic injuries reported a lower incidence of pneumonia following early tracheotomy.[1-3] Rodriguez and colleagues[1] randomized 106 trauma patients to early (Day 1-7) or late (later than Day 7) tracheotomy. In the early group, patients who underwent tracheotomy within 2 days of intubation had a 50% incidence of pneumonia versus an 85% incidence in patients who underwent tracheotomy after 3-7 days of intubation ($p < 0.05$). Lesnik and colleagues[2] reviewed charts of 101 trauma patients who underwent tracheotomy after 4 days or less versus after Day 4 of endotracheal intubation. The incidence of pneumonia was 19% in patients who underwent tracheotomy in less than 5 days versus 59% in patients who underwent tracheotomy at a later time ($p < 0.001$). Whisenant and colleagues[3] reported less severe respiratory tract infections in 77 trauma patients who underwent tracheotomy before Day 7 of endotracheal intubation versus after that time ($p < 0.05$). No differences were found with regard to age, injury severity score, or Glasgow Coma Scale score in any of these studies.[1-3]

In Summary.

- Both endotracheal intubation and tracheostomy increase the risk of aspiration and thus potential for airway colonization and nosocomial infection.
- Critically ill patients with a tracheostomy do not appear to be at greater risk for airway colonization and nosocomial pneumonia, independent of other variables.
- Early tracheotomy has been associated with a decreased incidence of pneumonia and/or less severe respiratory tract infections in patients with traumatic injuries.

The Case for Early Tracheotomy

Because no data clearly indicate that patients undergoing tracheotomy are at greater risk of surgical complications, airway damage, or nosocomial infection, it is perhaps more appropriate, as advocated by Bishop[5,33] and Heffner,[6,18,34] to base decision making regarding timing on an analysis of risks of the procedure versus anticipated benefits (Table 2).

Several studies provide compelling evidence that patients may wean more quickly from mechanical ventilation if managed with early tracheotomy. In a large series (n = 106) of trauma patients randomized to have tracheotomy performed on Day 7 or earlier versus 8 days or later, average (SD) days of mechanical ventilation were 12 (18) for the early group and 37 (3) for the late group ($p < 0.05$).[1] In a second study, patients who underwent tracheotomy within 4 days of intubation required 6.0 (3.4) days of mechanical ventilation prior to weaning compared to 20.6 (12.2) days in patients who received tracheotomy later ($p < 0.001$).[2] Other researchers have reported similar results.[3] Whether these findings apply to patients with other diagnoses is currently unknown.

As a guide to decision making, Heffner[6] advocates continued endotracheal intubation if extubation appears possible within 7-10 days of intubation (Fig. 2). If intubation is likely to be required beyond 21 days, early tracheotomy is performed. If duration of intubation is difficult to determine, daily evaluation is performed. At each step in the decision tree, consideration is given to both the risks and benefits of tracheotomy for that individual.

Decannulation

Techniques for decannulation include[35]

- progressively decreasing the size of the tracheostomy tube, then capping the tube, then decannulating if capping is tolerated,
- replacing a standard tracheostomy tube with a fenestrated tube,
- replacing the tracheostomy tube with a tracheal button, and
- combining use of a one-way valve with one of the above techniques.

Le and colleagues[35] reported equivalent success in achieving decannulation in patients (n = 10) randomized to conventional capping during weaning from trachoestomy or use of a one-way valve; 5 patients were successfully decannulated in each group. However, median time for decannulation was 18 days (range 14-21 days) with the one-way valve and 23 days (range 8-36 days) with standard capping. Also, patients with the one-way valve appeared more comfortable.

The use of the capped fenestrated tube is another option. Trials are initiated with the patient breathing through the tube with the cuff deflated. When the patient tolerates this procedure without distress, the tube is removed. If the airway can be protected, an uncuffed tube should be used because the presence of the cuff, even when deflated, can increase airway

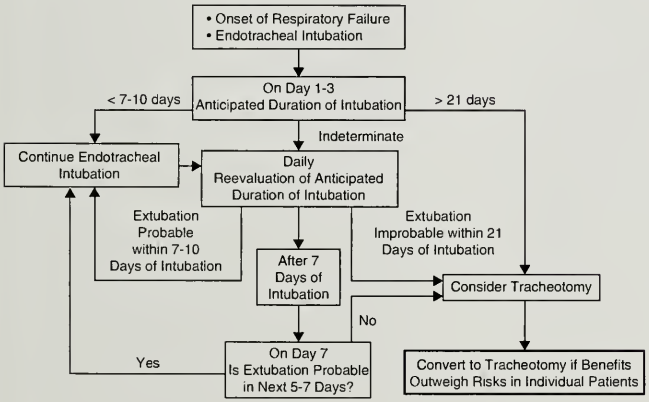

Fig. 2. An algorithmic approach to the timing of tracheotomy in patients who are intubated and mechanically ventilated for respiratory failure. (Reproduced, with permission, from Reference 6.)

resistance sufficiently to prevent successful decannulation.[36]

The tracheal button maintains the stoma and allows the patient to breathe and clear secretions through the upper airway. The patient can be suctioned or ventilated through the button if the cap is removed. When the patient can clear secretions and breathe without support, the button is removed and the stoma allowed to close. The button should be rotated daily to prevent tracheal tissue from adhering to the device and facilitate removal when this becomes possible.[37] In a study of 163 surgical and medical patients who used the Olympic Trach Button (Olympic Medical Corporation, Seattle WA), 92% achieved decannulation within 4.2 days (range 3-5 days). The remaining subjects were sent home with the button in place and decannulation occurred at home.[38]

Prior to decannulation, the trachea should be examined to determine whether obstructing lesions are present. In 81 brain-injured patients examined prior to decannulation, Law and colleagues[39] reported that 67% had obstructive airway lesions. Complications included tracheal granuloma (56%), tracheomalacia (23%), and tracheal stenosis (12%). No correlation was found between presence of these lesions and age, diagnosis, days of intubation, or duration of tracheostomy (mean [SD] 4.9 [3.5] months, range 1-23 months). The identification of granulomas is imperative because they can cause substantial airway obstruction and therefore prevent successful decannulation. Further, tracheal granulomas can usually be removed via laser therapy.[39]

Summary and Recommendations

As of this writing, there is no consensus with regard to the timing of tracheotomy. Although data exist that support early tracheotomy, the alternative viewpoint is supported as well because of the high incidence of morbid consequences with prolonged tracheostomy. Clearly there are institutional and regional variations in beliefs about optimal timing of tracheotomy. It is important to consider the individual patient when making this decision. The prudent clinician will regard all the evidence with respect to the individual patient's prognosis and needs, considering both the risks and benefits of early versus late tracheotomy.

The Consensus Conference on Artificial Airways in Patients Receiving Mechanical Ventilation[40] held in 1989 stated, "Existent deficiencies in our understanding motivate the need for further research. These include studies that

- quantify issues of comfort, psychologic benefit, and nursing care as they relate to an endotracheal tube or tracheostomy,
- develop a reliable scoring system that would predict a given patient's need for continued airway support,
- determine the incidence of nosocomial infection after tracheotomy, and
- prospectively quantify tracheostomy-related infection of the injured larynx."

These directives, now 5 years old, are no less timely and far more urgent than when they were originally written.

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Hoffman Discussion

Bach: We have an article in *Chest* reporting that people who had used both trach and nontrach positive pressure ventilation preferred the latter.[1] My question is, What about the patients who receive transtracheal oxygen? Has anybody asked them if they prefer that to nasal catheters or an Oxy-Frame system? About the patients with restrictive disease who are receiving the oxygen—who were you talking about?

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Hoffman: In regard to the first question, if you look at the series that are reported in the literature,[1-3] as well as my experience,[4] the frequency of discontinuing transtracheal oxygen runs between 8 and 30%. Smaller series tend to have more patients who discontinue transtracheal oxygen use, compared to larger series. We have had some patients who wanted to discontinue it—probably about 10%—even though we carefully select our candidates. I think some of the variations in discontinuing transtracheal delivery relate to how carefully candidates are selected and whether they have realistic expectations about what it will do.

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Bach: Well, how many people discontinue nasal cannula oxygen? It's not just discontinuing it. There's an element of cognitive dissonance there. If they've already got a tube in their necks, they might as well hook themselves up to it, but if you ask them in retrospect, which did you prefer. . . .?

Hoffman: If you ask them in retrospect, the ones who continue to use it say that they would like to continue using transtracheal. There are some people who pull their catheters out, like the 8-10% or the 30% in the larger series. We've had patients who have pulled their catheters out. I mean, it's very easy to discontinue. You just take it out, put on your nasal cannula, and go on your way. Some people do that.

Bach: How many people discontinue nasal cannula oxygen or just refuse oxygen period?

Fletcher: They don't comply. They say they do.

Hoffman: They don't comply. When they come in to see you they have their cannulas on, but what do they do when they go home? I don't know how many people refuse to use it at all at home.

Bach: So, it's really hard to say which is better tolerated by the patient, isn't it?

Fletcher: I think it's not a matter of tolerance as much as it is compliance, or adherence. Now, granted, transtracheal patients are much more highly selected. They tend to be better compliers. Maybe that's because you selected them because you knew they were going to be better compliers. I don't know. But, they tend to use their oxygen close to 24 hours a day, whereas the cannula patients don't necessarily take it off and throw it away, but they don't use it. They come to visit me in clinic, and they don't have their oxygen with them. "Are you on home oxygen?" "Oh! Yeah!" "Well, why don't you have it with you today?" "Well, 'cause I was coming to see you." So, it's obvious that they don't—a lot of them don't comply.

Kacmarek: Just to follow up on that comment, Can the studies that you referred to regarding transtracheal oxygen therapy versus nasal cannula and your results on the 12-minute walk and on the admission rates be explained by compliance? Did you take into consideration how well the nasal cannula patients complied with oxygen therapy use, as compared to those patients who had transtracheal catheters? Is there really a difference in device or is the difference purely a result of one group complying with a device and another group not complying with their device? Is that why we see these differences?

Pierson: Bloom did look at that.[1] The Bloom study did show that the people with the transtracheal used their oxygen a lot more than the people without transtracheal.

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Hoffman: We have not measured compliance, or adherence, directly by counting the hours of oxygen use in any of our studies. We tried to talk an

oxygen company into developing a device that could be placed in-line with the oxygen delivery system and monitor whether the patient was using oxygen—something that would pulse with inspiration and expiration and thus monitor actual use. It's very difficult to monitor compliance. You can put a timer on a stationary system, but then what about the portable delivery device? You can record the number of hours that patients use the stationary system with either nasal or transtracheal delivery, but you don't know if the tubing is lying on the couch or whether it's connected to the patient. Our feeling was that to answer this question objectively, we needed something in-line, some kind of counter, that would cycle when the patient was breathing so there would be no question that oxygen was being used. But, we weren't able to convince the company to develop such a monitoring device.

Celli: I have two comments, one in relationship to oxygen. In my opinion, our main goal should be compliance. If you had an ideal way to do it, the ideal would be transtracheal, 24 hours a day, guaranteed to go where it should go and with a beneficial effect. I think the problem for not placing more transtracheal oxygen is what the cost is when somebody's got to clean it every 3 weeks, see the patient, make sure that if it comes out the patient reports that and you get it back in, and the second is the issue of whether patients will, indeed, support the maintenance and education that you require for that. So we're selecting a group. But if it were the ideal world, I think it makes a lot more sense to use transtracheal than any other form of oxygen conservation device. Let me make a comment now on tracheostomy. The problem is that we don't know the epidemiology of tracheostomy. We're submitting to ATS an abstract on 800 prospective admitted patients to 4 ICUs in order to determine what is the practice of tracheostomy. I think John Hefner was right.[1] The mean intubation time of people in the medical intensive care unit (nonsurgical) is 8.2 days ± 7 in our series. If you do risk analysis in this data, the point at which the curve ceases to be significantly different on the next day is 17 days. Right now, what people are doing is what John (Heffner) is recommending. People keep a patient intubated and when it's nearing a couple of weeks, they go ahead and trach them. From that pattern, it would appear that what they're doing is the appropriate thing.

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Hill: I'd like to expand on your comments about face-mask or nasal-mask ventilation as an alternative to tracheostomy. I'm a strong proponent of noninvasive ventilation, but I think in the settings in which most patients are undergoing tracheotomies, it has to be used with a lot of caution. We need more information before we can see it as really a safe alternative, except in selected cases. The reason is that many patients are coming out of acute exacerbations of their disease, as you're well aware, and have problems with secretions and swallowing. In our respiratory care unit, when we're trying to decannulate patients, we're often dealing with secretion and swallowing problems that are not handled well by nasal- or face-mask ventilation. So, I think we need to offer a caveat that these patients be managed very carefully if noninvasive ventilation is to be considered.

Hoffman: I agree with you totally. I think you're looking at a small group, perhaps a select group, and certainly not everyone can be managed in that manner. To go back to the second part of Dr Bach's question, what type of patients with restrictive disease are being managed on oxygen? The most common diagnosis, in my experience, is interstitial pulmonary fibrosis. We see a lot of patients with that diagnosis in Pittsburgh, perhaps more because of our active lung transplant program. In my experience, and I didn't include this in the talk, many of those patients, although not all, do not make good candidates for transtracheal oxygen delivery. We tend to get people referred when they are on 6 liters of oxygen and can't be oxygenated. The response is "let's put in a transtracheal catheter." In some instances, they've been on a face mask, and they don't want that; again, the response is "let's put in a transtracheal catheter." There is no question that you can use transtracheal delivery for such patients. The more appropriate question is should you do it? We've managed patients who have used transtracheal delivery at high flows without problems, including 1 patient who simultaneously used both nasal cannula delivery at 6 L/min and transtracheal delivery at 6 L/min for 12 months. But, this is not typical. Many patients who require high-flow oxygen have a rapidly progressive disease. I believe you have to carefully evaluate what might transpire in their last months of life. If they are rapidly going downhill, they may not have 2 good months to get beyond the immature tract time. If you use transtracheal in such patients, you risk having a very bad experience, not only from the healthcare provider's standpoint but also from the patient's standpoint.

Make: We talked about some of the selection criteria—patient willingness to be taught and perform the extra care and maintenance. There's another selection criteria, and that is patient willingness to have the procedure done in the first place. Maybe I'm doing something wrong, but I offer my patients the option of a lot of different kinds of devices when they're using oxygen, including con-

serving devices and transtracheal. I must admit it's uncommon for them to jump up and down and say, "Yeah, I want transtracheal oxygen," no matter how you explain it to them. I think it's very difficult to compare transtracheal oxygen to other types of oxygen delivery because of those selection issues.

Hoffman: I have heard a lot of other people say exactly the same thing, and that was also true for us until we started having more people on that delivery system. A lot of the patients that we managed were at the VA. They were in and out of clinics, seeing each other frequently. Under those circumstances it was a lot easier to recruit people for our study because a lot of people were using transtracheal oxygen. But for the patients coming in from home, not seeing other patients, it's something very new and different. Yes, I think it can be quite difficult to persuade them that this is something they really want to do, even though you think it might be good for them.

Leger: What is the proportion of patients on oxygen conserving devices especially transtracheal in the United States? Do you have an idea?

Hoffman: That I don't know. I can only speak from the perspective of patients we follow in Pittsburgh. We have no patients on the pendant. We have probably 1% or less of our patients using a reservoir cannula and about 10% on transtracheal delivery. In the Pittsburgh area, a lot of patients use liquid systems, and demand delivery is rarely used. However, from talking with others, some areas of the USA appear to have many more patients who use demand delivery systems, especially portable systems, with a concentrater as the stationary. Another alternative is a portable liquid system plus a concentrater, if the home care company will do that because it reduces overall cost.

Pierson: My comment is on the numbers of patients. Not knowing how many patients are on home oxygen in the United States is an embarrassment and a problem for those of us who want to work in that area. Likewise, we really have no good idea how many patients are on transtracheal or who use conserving devices. We don't even know how many patients we have on concentraters and liquid oxygen. I recently participated in an international symposium on home oxygen therapy in Japan. Although the United States has had more experience with this modality of therapy than all of the other countries in the world combined, ours was almost the only country for which no figures could be given on how many patients we had, and by what techniques, and the other demographics. According to Tom Petty, the estimate that has been given by the American Lung Association is about 800,000 patients in the U.S. on home oxygen therapy. That is probably pretty close to correct; at least I believe authorities believe it's nearly correct. I have heard estimates somewhere in the range of 10-15,000 patients on transtracheal oxygen, and that could be off by a factor of 2, I suppose; but even so, that approaches your 1-2% estimate, at least for transtracheal oxygen. I think with regard to the conserving devices, the variation regionally is so huge, with some regions of the country using them almost not at all, and others using them much more often, related in part to what can be reimbursed by their carriers in different regions. It's very hard to get a handle on that.

Hoffman: That's correct. Certainly in the USA usage is very much influenced by reimbursement.

Leger: I would like to comment on the long-term complications of tracheostomy. In your talk, you spoke of the constant care needed and the immediate complications of the tracheostomy. I would like to add some information. We have had complications arise after 5 or 10 years of tracheostomy and long-term ventilation. One problem has been maladaptation of the tube to the trachea causing pain, bleeding, and granulomas. This problem has mainly been seen in patients with scoliosis, which over the years evolves and increases in curvature. Another interesting aspect that has been very new and challenging for me is the higher incidence of complications we have observed in neuromuscular patients who need tracheostomy after many years of noninvasive ventilation. These complications are similar to what we see after prolonged intubation. These complications are very difficult to manage and in my opinion need to be further studied.

Dunne: I just want to comment because the purpose of this conference is to identify controversies. Not all demand systems are created equal. Part of the confusion is that we have different, competing technologies, in terms of what is the ideal demand system. Part of the problem that we face is that the incentives for the development and standardization of this technology—specifically who will pay for it—has pretty much taken some of the wind out of the sails of the manufacturers who are trying to arrive at a common frame of reference. Should the system be totally and purely on demand, with a timing mechanism that instantly autoadjusts to each inspiratory breath or can a preset feature utilizing some variability of breaths suffice? So, when we start looking at demand systems, we have to realize that some of them work differently from others, and, consequently, performance and outcome may be skewed. Quite honestly, I don't see the manufacturers of these devices rushing toward putting a lot of R&D dollars into them until there is some stability, in terms of the marketplace and who is ultimately going to pay for them.

Hoffman: I agree with you and that is why I tried to highlight the study by Braun and colleagues.[1] This reference is one of the few, in fact the only one I found, that compared outcomes in the same patients while using different demand delivery devices. All systems were not the same—and all were not the same in each patient. If the physician has to do exercise testing with several systems to learn which prevents desaturation, this creates a lot of problems. If it were clearer that patients who are predominantly mouth breathers, nose breathers, or pursed-lip breathers, or had different types of pulmonary disease did better on one system or another, decision making would be easier. We need more comparative studies. Maybe clinicians who more frequently prescribe demand delivery systems have more definitive answers, although they have not been published. But it appears that, at least from the results of this study,[1] there is no one device that always works well.

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Stoller: I have one comment with regard to the learning effect with the transtracheal technique. We were struck by the prevalence of low-grade complications in our earlier experience.[1] Dr Navdeep Rai, one of our fellows, Kay Stelmach, and others in our group got interested in looking at our own in-house personal experience with regard to a "learning effect" in inserting transtracheal catheters.[2] In stratifying complications by (1) actual complications related to the insertion itself versus (2) complications developing later (such as infection, misplaced catheters, and mucus balls), it appears that there is little demonstrable training effect in our hands. There may be a small learning effect for techniques related to the actual insertion, but the longer-term problems of mucus balls, cleaning, infection, chondritis, and misplaced catheters, do not seem to decrease with increasing experience. If you think about it, this is perfectly logical because these long-term problems have nothing to do with operator expertise. It has to do with the technology and the equipment itself. I wonder: (a) Has this also been your experience? (b) For the sake of the Journal's readers, I think it would be helpful, perhaps, to have you tell us exactly what indications and screening criteria you apply because your experience is certainly exemplary, in terms of low complications of transtracheal oxygen.

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Hoffman: Our experience has been the same as yours in regard to a decreasing number of procedural-technical complications over time. In 1987, we published an article in the *American Review*,[1] in which we had substantially more procedural complications than we had in our latter studies.[2,3] I think the critical elements are the teaching program, monitoring, and follow-up. We learned how to do this more efficiently with more experience. In regard to the complication of mucus balls, we initiated a program of calling patients at periodic intervals to monitor their progress and assess if they had problems. We did this because we believed the patients might not call us until a problem was more severe or might not pick up small changes on their own. We've also emphasized that we were available for problem-solving. During our studies, the nurses who provided care certainly did put in a lot of hours. I think their dedication made a big difference. We were personally very committed to the success of this project. I'm certain some of this translated to the patients—perhaps making them more receptive to the teaching and helped with problem-solving.

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Center-Based vs Patient-Based Diagnosis and Therapy of Sleep-Related Respiratory Disorders and the Role of the Respiratory Care Practitioner

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I. Introduction
II. Review of the Literature
III. Center-Based Polysomnography vs Patient-Based Unattended Monitoring
IV. Who Is Trained for This New Field?
V. Therapy for Sleep-Disordered Breathing
VI. What Is the Role of the RCP in Diagnosis and Management of Sleep-Related Respiratory Disorders in the Home?
VII. Summary & Recommendations

Introduction

Sleep is an active brain process during which biochemical and neuronal changes occur that are distinctly different from those of wakefulness. In health, sleep results in a restorative or refreshed sense of well being. In some people, sleep is accompanied by profound changes in ventilation and gas exchange that interrupt sleep and/or diminish its restorative effect. In patients already compromised by cardiopulmonary disease, sleep is an additional stressor that may initiate or potentiate clinical deterioration. Recent research indicates that sleep disorders are prevalent in the general population, being present in approximately 18 million people in the U.S.[1-3] Consequently, physicians, researchers, and many allied healthcare professionals have become involved in understanding and modifying the impact of sleep on disease.

The gold standard for documenting a sleep-related respiratory disorder is polysomnography performed in a hospital-, or center-based laboratory and supervised by technical personnel (see Appendix). A single study lasts 6-8 hours, produces approximately 900 pages of data, and is manually scored and interpreted. An alternative approach is the use of portable equipment that can be taken into a patient's home and used for diagnostic or patient follow-up, with or without a technician in attendance. Although it is, in our opinion, biologically

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plausible to perform diagnostic testing and follow-up in the home,[4] this method is being strongly advocated[5] without substantial understanding of the costs, the sensitivity and specificity, and optimal training of technical assistants. In regard to the training of technical personnel, we believe that there is a role for respiratory care practitioners (RCPs) in the diagnosis and treatment of sleep-disordered breathing.

The purpose of this paper is to discuss differences and similarities between the diagnosis and management of sleep disorders in hospital centers (center-based) and in the patient's home (patient-based). We also discuss current treatments of sleep-related respiratory disorders, particularly the similarities and differences between continuous positive airway pressure (CPAP) and bilevel positive airway pressure (BLPAP). Finally, we propose a role—from diagnostic procedures to therapeutic management—for the respiratory care practitioner in the field of sleep medicine.

Review of the Literature

Our search of the literature primarily revealed publications focusing on the techniques and procedures of center-based studies (polysomnography and multiple sleep-latency tests), with some reports of technical validation of ambulatory and portable patient-based monitoring equipment. Technical validation of ambulatory and portable equipment has usually been accomplished by comparison to polysomnography during patient diagnostic studies. Few studies have sought to validate the technical feasibility and diagnostic sensitivity of patient-based studies. Those studies that examined the usefulness of patient-based sleep monitoring systems in clinical management did so in a case-study or clinical-cohort format with relatively small sample sizes, and, therefore, may not be generalizable to larger populations. Consequently, no studies have determined the clinical impact of patient-based diagnostics and therapeutics for sleep-disordered breathing on healthcare.[6]

Examples of questions that remain to be answered include, Do patient-based studies improve patient outcome? Is medical management based on patient-based study results more cost-effective or efficient than management based on center-based results? Is sufficient information collected with patient-based monitoring to facilitate diagnoses without additional studies? How does patient-based monitoring affect the clinical decision-making process. Until data are available to address these issues, the utility of patient-based monitoring for the management of sleep-related respiratory disorders will continue to be controversial.

For the purpose of this paper, the primary reference sources for definitions and diagnostic guidelines for sleep-related respiratory disorders are the American Thoracic Society's (ATS) Consensus Statement entitled Indications and Standards for Cardiopulmonary Sleep Studies,[7] and the American Sleep Disorders Association's (ASDA) Consensus Statement entitled Portable Recording in the Assessment of Obstructive Sleep Apnea.[8] Principles and Practice of Sleep Medicine (edited by M Kryger, T Roth, and WC Dement) is another principal information source.[9]

Center-Based Polysomnography vs Patient-Based Unattended Monitoring

For patients suspected of having sleep-related respiratory disorders, the standard diagnostic procedure at this writing (1994) is center-based attended polysomnography. During this procedure, while the patient is asleep, electroencephalographic (EEG) and cardiopulmonary variables are monitored under the direct observation of a technician. Because the focus of polysomnography is on monitoring during sleep (cardiopulmonary variables and other physiologic signals, eg, the tibialis electromyogram, or EMG, for periodic limb movement), the data recording montage is much different from that which is used during traditional EEG studies. Specific indications and contraindications for polysomnography have been developed to help one decide which patient is better studied by polysomnography than by electroencephalography, and these have been reviewed and published.[7,10]

As the recognition of and treatment for sleep-disordered breathing emerged, questions arose as to the necessity of complex, center-based polysomnography.[11] Alternative qualitative and quantitative evaluation methods for sleep-disordered breathing were proposed, and more limited monitoring was explored. These techniques range from simple ques-

tionnaire and oximetry studies and computerized analysis of tracheal breath sounds to monitoring analysis of respiratory movement, airflow, pulse, and oxygen saturation, during wakefulness and sleep.[12-15] However, until recently, no formally accepted criteria existed to describe and categorize the varying levels of complexity used for diagnostic procedures of sleep-disordered breathing. In an effort to describe these different levels, the ASDA has proposed criteria to differentiate and categorize different diagnostic approaches.[8] These have been modified and are presented in Table 1.

The four categories presented in Table 1 describe the current technologic approaches for diagnosis of sleep apnea. Because an overlap between categories may occur in the type and purpose of monitoring, one cannot assume that these categories are distinct in terms of complexity and/or cost. Is it correct to assume that an equal amount of time is required to score polysomnographic studies from 'healthy' individuals and from those with severe sleep apnea? Taken one step further, is there a difference, in terms of time and complexity, between scoring a normal polysomnographic study with 16 variables and scoring a markedly abnormal modified sleep apnea study with 4 variables?

Questions related to the length of time needed to collect, score, and interpret data collected from different diagnostic approaches need to be addressed. If clinical intervention is based upon a quantitative assessment of specific variables and if multiple variables can be measured and quantitated in the same amount of time as only a few variables (as may soon be feasible with microprocessor-based data acquisition and assisted scoring), and if the technician cost is similar between the two methods, then the argument that limited (or less complex) diagnostic studies are more cost-effective than polysomnography is considerably weakened. Therefore, until peer-reviewed studies that examine technician time required for patient setup, data scoring, and report generation have been completed, any cost differences between the previous types of diagnostic strategies remain speculative.

A number of portable and unattended recording systems[16] are now commercially available. Some systems are capable of recording and digitally storing the same variables as are hospital-based polysomnography systems, others record only select signals such as oxygen saturation, heart rate, chest-wall movement, and airflow. In general, good agreement exists regarding the ability of portable and unattended equipment to monitor and record information adequate to document the presence of apnea and other sleep-related respiratory disorders.[17-19] However, the diagnostic accuracy in unselected patients and the clinical impact of patient-based studies are still unclear. Insufficient evidence exists to indicate whether patient-based studies reduce the overall cost of management. A variety of nonrespiratory sleep disorders present with symptoms similar to sleep apnea syndrome and require polysomnography, following an inconclusive home-based study. For example, narcolepsy is one sleep disorder that causes hypersomnolence but has no respiratory component.

Table 1. Differences among Four Types of Sleep Apnea Monitoring*

Procedure	Technician in Attendance	EEG	EMG	EOG	EKG	Heart Rate	Respiratory Effort	Airflow	Body Position	S_{pO_2}
Center-based polysomnography	Y	Y	Y	Y	Y	Y/N	Y	Y	Y	Y
Comprehensive portable polysomnography	Y/N	Y	Y	Y	N	Y	Y/N	Y	Y/N	Y/N
Modified portable sleep-apnea testing	Y/N	N	N	Y	N	Y	Y	Y	Y/N	Y
Continuous bioparameter recording	Y/N	Y/N	Y/N	Y/N	Y/N	Y/N	Y/N	Y/N	Y/N	Y/N

*EEG = electroencephalographic activity; EMG = electromyographic activity; EOG = electroocular activity; EKG = electrocardiogram; respiratory effort = abdominal and thoracic chest-wall movement; airflow = nasal and/or oral airflow; body position = right, left, or supine, or semi-Fowler's position; S_{pO_2} = oxygen saturation measured with pulse oximetry.

Unlike the consensus statements and technologic standards available for hospital-based polysomnography, no such consensus statements and standards exist for patient-based studies done in the home. As mentioned in the literature review, studies to date support only the technical feasibility of patient-based studies.[20] Evidence is inadequate to indicate how information obtained from patient-based studies is useful in the clinical decision-making process for the diagnosis and treatment of sleep-related respiratory disorders.

Regardless of the range of available technology, physician opinion regarding patient-based monitoring may also be influenced by cultural issues. For example, we have observed that home monitoring for sleep apnea appears to be more popular in European countries than in America. This may be due in part to the lack of standardized sleep laboratories in Europe, which, in turn promotes the need for alternative methods to diagnose or, at least, to screen for, sleep-disordered breathing. Because the patient-based monitoring methodologies adopted by the Europeans seem to adequately satisfy some of their needs, the difficulty behind American acceptance of patient-based studies may be based, at least in part, on reasons other than the accuracy of the instrumentation. Therefore, much work needs to be done to examine healthcare delivery issues related to home-based studies.

Who Is Trained for This New Field?

Although center-based polysomnography is governed by rules for technician credentialing and equipment sensitivity and specificity,[7,8] no formal rules exist for performing patient-based studies of suspected sleep-related respiratory disorders. We believe that this lack of formal definitions and personnel credentialing has resulted in a void often filled by marketing rather than clinical practice concerns. We summarize the suggestions for personnel qualifications and operational guidelines for patient-based studies that have been developed by the ASDA in an effort to develop a professional model for those performing patient-based studies.[8]

- An order from a licensed physician is required to initiate a portable sleep apnea recording.
- Only an appropriately trained technologist, physician, or other doctoral-level professional should conduct a portable sleep apnea recording.
- Only a physician or other doctoral-level professional with satisfactory training in sleep medicine and adequate experience in the interpretation of standard polysomnograms should interpret portable recordings.
- An interpretation and a representative sample of the record must be provided to the referring clinician; the complete recording (with full data disclosure) must be available upon request.
- Physicians should not order and commercial companies should not conduct portable sleep studies for obstructive sleep apnea syndrome when conflicts of interest exist.

These ASDA recommendations address general and, perhaps, noncontroversial policy issues. We wish to discuss more specifically the role of "an appropriately trained technologist." During center-based studies, the technician's scope of practice involves monitoring and recording the patient's physiologic variables, observing for acute changes in clinical condition, and relaying pertinent information to the appropriate house staff.[9] In addition to this, the technician also monitors the quality of the recorded data and performs any maintenance to the equipment that may be needed during the study (eg, recalibration of physiologic amplifiers or reattachment of displaced sensors). With regard to patient-based studies, the technician's scope of practice is different. If portable monitoring is performed as an attended study, then the technician's role is similar to that in center-based polysomnography. Alternatively, if it is an unattended or ambulatory study, the technician's role may be as simple as dropping off and picking up the equipment at the patient's home.

The technician's broad scope of practice for patient-based studies and the 'generous' wording used for personnel qualifications make a more detailed definition of what constitutes an "appropriately trained technologist" desirable. For example, should an identical level of training be required for all tech-

nicians who perform center-based and patient-based studies? Distinctly different skills are needed to perform full or comprehensive polysomnography as opposed to 4-channel modified sleep apnea testing. Therefore, would an appropriately trained technologist be one who possesses the knowledge base and skills to perform any diagnostic study (regardless of complexity) or only those at which he could be judged to be proficient? With the limited approach, how would proficiency be determined? Clearly a need exists for recognized and accepted standard criteria to describe a technologist's level of skill.

Therapy for Sleep-Disordered Breathing

Once a diagnosis of sleep-disordered breathing has been confirmed, a decision to institute some form of therapy is made. Even though many forms of therapy for sleep-disordered breathing have been explored,[19] those therapies that require technical help are CPAP and BLPAP.

CPAP

CPAP devices deliver a constant pressure throughout the airway during the spontaneous respiratory cycle. The constant pressure acts as a pneumatic splint to prevent upper airway collapse.[9] The ATS statement[22] suggests the following indications for CPAP therapy: (1) CPAP is indicated in the treatment of patients with clinical obstructive sleep apnea manifested by neurocognitive dysfunction; (2) CPAP is indicated in the treatment of patients with clinical Cheyne-Stokes respiration or central sleep apnea manifested by either neurocognitive dysfunction, daytime hypersomnolence, or cardiopulmonary dysfunction; and (3) CPAP is not indicated in patients with simple snoring.

BLPAP (eg, BiPAP™)

Like CPAP, BLPAP devices are designed to treat sleep apnea. The difference between CPAP and BLPAP is that CPAP delivers a constant inspiratory and expiratory pressure, whereas BLPAP can deliver one level of positive airway pressure during the inspiratory cycle and a lower level during the expiratory cycle (that is, BLPAP devices mimic simple ventilators). However, no published controlled trials have established any advantages to using the more costly BLPAP[8] instead of CPAP.

We submit that the issues related to center-based vs patient-based diagnostics are also relevant to therapeutic issues. Center-based approaches are currently used to set up and titrate CPAP and BLPAP systems. Newer servo-controlled machines can monitor pressure waveforms and independently adjust settings to patients' needs. This allows such therapy to be initiated in the home or to be updated without repeated center-based studies. Therefore, a definition of the goals, outcome, and personnel qualifications for this approach is needed.

What Is the Role of the RCP in Diagnosis and Management of Sleep-Related Respiratory Disorders in the Home?

Due to the increased recognition of sleep-related respiratory disorders in our society and the large number of people needing assessment and treatment, we believe that opportunities exist for more RCPs to become involved in sleep medicine. Many practitioners already play an integral role in diagnostics, therapeutic intervention, and long-term management of patients with cardiopulmonary disorders. These skills can be expanded to include working with the patient with sleep-disordered breathing.

Because RCPs receive basic training in pulmonary physiology and have the ability to collect and determine the quality of data that reflect ventilatory status (eg, arterial blood gas measurements, end-tidal CO_2 values, pulse oximetry data) and possess an understanding of and level of expertise in airway and ventilator management, they may already possess the expertise needed to effectively initiate and titrate CPAP or BLPAP therapy. However, we believe that for RCPs to advance their scope of practice to include the broad field of sleep medicine, additional training is needed.

To enable RCPs to move into full participation in sleep medicine, a formal curriculum should be developed. This curriculum should provide training in basic sleep physiology (eg, the various states of sleep, circadian rhythms, and chronobiology) and an understanding of nonrespiratory sleep disorders such as narcolepsy. Additionally, practitioners should become familiar with performing polysom-

nography and learn to score the data acquired during the study. Finally, once these skills are acquired, with some additional training, RCPs could become credentialed as registered polysomnographic technologists (RPsgT).

Summary and Recommendations

Unlike center-based laboratories that follow prescribed technical standards, patient-based studies lack technical standardization. Whereas center-based studies are usually performed by technicians who have met certain professional criteria, patient-based studies can be performed by anyone, even those without clinical training. Although guidelines are currently being developed to direct the use of unattended monitoring in the home, a need exists for specific guidelines that delineate professional qualifications for those who initiate and titrate therapy for patients with sleep-related respiratory disorders.

The opportunity exists for respiratory care practitioners to become involved with creating guidelines for patient monitoring and therapeutic intervention, in both center-based and patient-based settings. A need exists for multicenter, peer-reviewed, controlled studies to establish standard recording variables and machine specifications for unattended monitoring devices. Also, the role and cost-benefit ratio of unattended monitoring in healthcare delivery need to be defined. Therefore, with the demand for technology-oriented research, clinical outcome studies, and professional standards, more respiratory care practitioners have the opportunity to expand their practice into the field of sleep medicine.

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APPENDIX

Glossary of Terms

The following terms are used in this document in a specific way. The purpose of the Glossary is to provide a standard terminology for the different techniques used to study patients suspected of suffering from sleep-related respiratory disorders.[6]

Standard polysomnography: Polysomnography is a diagnostic study used in the evaluation of sleep apnea. It is performed under technical supervision, in an appropriate medical setting, and includes electromyography, electrooculography, electroencephalography, and airflow, respiratory effort, oxygen saturation, electrocardiography, and body position monitoring. Limb-movement, vocalization, and end-tidal P_{CO_2} monitoring are optional.

Screening somnography: Screening studies are conducted on individuals who are asymptomatic (although possibly at risk) for obstructive sleep apnea. These studies are reserved for epidemiology and/or research protocols. When a patient presents to a physician for evaluation, any study done, regardless of the complexity, is considered a diagnostic procedure rather than a screening test.

Portable recording: Portable recording uses movable equipment that is easily transported, for use outside of a center-based laboratory.

Ambulatory recording: Ambulatory recording is conducted in a manner that allows a patient to walk around and engage in other usual daily activities during data collection. If portable equipment is used, the patient may be connected to stationary equipment by direct (modem) or radio (telemetry) linkage.

Attended monitoring: During attended monitoring, trained personnel are physically present and able to intervene, throughout the recording session (data observed via modem link is not considered attended).

Unattended monitoring: During unattended monitoring, trained personnel are not physically present throughout the recording session. Unattended studies include those in which a patient is connected to a physiologic data recorder by a trained technician. The technician then leaves the area until the study is concluded. During the technician's absence, neither the patient nor the recording equipment is under any type of supervision that makes direct intervention practical (data observed via modem link is still considered unattended). In some cases, patients independently perform their own sensor application and machine hook-up and initiate and conclude the study.

Decker Discussion

Kacmarek: You talked about four levels of evaluation, but one thing you didn't explain is how you decide which level is appropriate for which patient. When you consider the delay in getting into a sleep lab—at least in our institution—it's 3 months or longer. The patients can die before you can get them in the lab. How do you make a decision on which patient can be handled at each different level of evaluation?

Decker: At the present time, the technical level of evaluation is determined by the physician to whom the patient presents. Patients with sleep-disordered breathing have a variety of symptoms; therefore, they may present to a psychiatrist, a neurologist, or a pulmonary physician. Each specialty has its own variables to examine; whereas, a neurologist prefers to diagnose sleep apnea with technology utilizing EEGs, a pulmonary physician may be more interested in respiratory variables. Consequently, the level of study performed is going to depend upon the specialist to whom the patient presents, as well as the pretest probability for apnea.

Pierson: My question is related to Bob's. I've been troubled, as a non-sleep person, by a kind of cognitive dissonance between the standards set by the recommendations of the American Society for Sleep Disorders (ASDA) and what I see as the actual logistics of the problem. If 4% of the working population has this disorder, and if the only approved diagnostic maneuver is a formal, center-based, 2-night polysomnographic study, a fair number of that 4% will never live long enough to get their appointment—because there's a huge difference between the numbers of people for whom this is recommended and our capabilities for actually carrying out these tests. Do you know how many sleep labs there are and any kind of figures relating to how many studies per month or per year, can be carried out in per million population, that sort of thing?

Decker: There are approximately 225 ASDA-certified labs and approximately 2,000 other labs. This would still be inadequate.[1]

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1. National Commission on Sleep Disorders Research. Wake up America: a national sleep alert. Washington DC: Government Printing Office, 1993.

Fletcher: The American Thoracic Society will publish, probably in about 6 months, a consensus statement on this exact issue.[1] I think it's much more liberal than the ASDA's statements. In patients in whom you strongly suspect sleep apnea (even if there is a certified polysomnography lab right next door), it's OK to go ahead and do home monitoring because there will be a very low incidence of false positives and a very high sensitivity. That patient can then be titrated on CPAP with a nap study, provided the nap study includes a period of REM sleep. At the other end of the spectrum are patients in whom you don't really suspect apnea but who have been referred to you because their wives said they snored very loudly and they have high blood pressure, perhaps a negative home-based monitoring study will be adequate to keep them out of the lab. Everybody else in between—the patient who snores and has reported cessation of breathing—should be studied in the sleep lab because even if you screen them with home monitoring and it's a negative study, you still haven't ruled out sleep apnea. So, they're going to have to come to a sleep lab anyhow, and all you've done is added to the cost. Second point, you have to be careful about home monitoring. At least in Houston, before I left, there were home care companies monitoring at home for $600 a pop. A sleep study in a lab would cost $800. So, for the $200 difference, you really lost a whole lot. The third and final comment is that I have a problem with pulmonologists who start cardiopulmonary sleep labs and do sleep studies and send reports back to you that say this patient doesn't have sleep apnea. You're still left with a sleepy patient. Why? In fact, I got one of those reports back last week from an ASDA-accredited sleep lab run by a pulmonologist. The report said the patient doesn't have sleep apnea. Here's a guy off work because of hypersomnolence. So, I think you need to be a full service sleep lab in order to be cost-effective for the patient and the insurance company—you've got to do more. You've got to rule out narcolepsy and a whole bunch of other things. You can't just send back a report and say no sleep apnea present. The one limitation in that respect is this "new syndrome" of Guilleminault, upper airway resistance syndrome. That probably eventually is going to result in the necessity for monitoring esophageal pressures, which, of course, can't be done with home monitoring. I think that's probably a very valid question—unanswered at this point, but still a valid question.

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Decker: In response to that, I agree with most of what you said, especially the statement that 20% of the people do have more than one sleep-related abnormality. A study that uses comprehensive portable polysomnography, including a small machine that will record electroencephalographic activity in the patient's home, will give you that information; so, I don't know if it's correct to say that portable monitoring won't give you the correct information. It's based upon the approach that you use.

Fletcher: Is that cheaper, and can as many be done in one night by one technician as in a centralized sleep lab?

Decker: We don't have that information. There have been no studies in the literature to describe the cost impact or the diagnostic accuracy of that approach.

Hill: In response to your description of the respiratory care practitioner's role, in my area of practice, anyway, respiratory care practitioners are doing exactly what you described. The home respiratory care companies take the patient out of the lab, set up the equipment in the home, and do a lot of monitoring, follow-up, and maintenance of equipment. Isn't that happening all over?

Decker: We've seen that in our area. It brings us back to an earlier discussion of the level of expertise needed for respiratory care practitioners? What we're proposing is that at the academic level, respiratory care practitioners be introduced to sleep and demonstrate a fundamental understanding. I don't think a respiratory care practitioner can just walk out of school or off the hospital floor, go to a patient's home, and perform a sleep study or set up CPAP. There is a need to define some type of advanced training that is not available in the current curriculum.

Hill: Well, they certainly do it, and they're getting on-the-job training where I am. We've found that as long as respiratory care companies have experienced practitioners, they provide good service. We also find that modified portable sleep-apnea testing for follow-up monitoring in the home (such as with the Edentec monitor) is very useful for determining persistence of desaturations, apneas, and the like. Our problem is that we can't get them reimbursed.

Decker: Right. That's a problem, but there is no defined role or advantage for follow-up in an asymptomatic patient. A study needs to be done to examine these issues.

Hill: The companies are doing them for free. They can certainly can be done for less money than center-based polysomnography, but they are not being reimbursed at all because there's a conflict of interest if the company that's providing the equipment does the monitoring (ie, they might "doctor" the results). It's a problem.

Sortor-Leger: I agree with Dr Hill in the value of home monitoring for patient follow-up and would like to make a few comments. First, in response to the problem of reimbursement, one suggestion I can give is to create an outpatient service through the hospital for home monitoring. In Dallas, we had very little difficulty with reimbursement when we provided the service in this manner. My second comment is in regard to the training of respiratory therapists to do sleep monitoring. In my experience, most therapists have no difficulty managing technical aspects of monitoring but more complicated and less often mastered are the integration, interpretation, and management of the clinical aspects that present during monitoring (eg, air leaks and obstruction). I'm not sure what additional education is needed to improve these thinking skills, but it is in this area that I think our educational system needs to focus.

Bach: There is discomfort with commercially available nasal interfaces. You mentioned that. But you didn't mention custom interfaces. There are three kits for three different styles of nose piece that are custom molded to the patient. In general, they are very well tolerated. Also, we make our own nasal interface. A prosthodontist makes a plaster moulage of the patient's nose, and we make a transparent, low-profile acrylic nasal interface. These are really comfortable alternatives.

Kacmarek: I want to respond to John's statement (Bach's). A lot of the problems with nasal interfaces, even if we use the commercially available ones, relate to inappropriate sizing and inappropriate instruction on how to secure the device. If the proper size device has been selected for the given patient, and the patient has been taught that the mask does not have to be ratcheted to obtain a seal, I've rarely had difficulty with sizing or with a seal when using commercially available masks. I think it's an extremely rare situation that requires a custom mask.

Bach: The higher the pressures you use, though, the more likely you are to have those problems.

Kacmarek: How high a CPAP pressure are you using?

Bach: Well, we use these mostly for patients requiring noninvasive ventilation. So, it is different, but when you do use higher pressures, even with CPAP, they can be helpful. It's more comfortable for the patient to have a custom-molded nose piece. The second thing is that I've seen patients with obesity hypoventilation syndrome who have been diagnosed as having severe central and obstructive apneas. They were severely hypercapnic and were treated with CPAP, and then with low levels of BiPAP,™ to absolutely no avail. Then they were told they had to use oxygen or be trached. We have been able to successfully manage many with mouthpiece or nasal ventilation. I understand the theoretical benefits of having a little bit of EPAP. But, some patients really may not need the EPAP so much as a large IPAP/EPAP pressure difference or nasal ventilation to assist inspiratory muscle function.

Decker: No, that won't work with obstructive sleep apnea. The study done by Mark Sanders[1] pointed out that apnea or upper airway occlusion can happen on expiration as well as inspiration. So, you're still going to need a positive expiratory pressure.

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1. Sanders MH, Kern N. Obstructive sleep apnea treated by independently adjusted inspiratory and expiratory positive airway pressures via nasal mask: physiologic and clinical indications. Chest 1990;98(2):317-324.

Bach: I know in theory that's the case, but did he really try just using high IPAP? Or a high IPAP/EPAP difference? I'm talking about 20+ cm H_2O, the kind of IPAPs that I use to ventilate my patients. I think you shouldn't assume it won't work—because I have managed some very severely affected obesity hypoventilators with sleep-disordered breathing by nasal ventilation, who could not be helped by CPAP or BiPAP.™ Also, if you've got a patient who is extremely symptomatic and you just do simple nocturnal oximetry and find many desaturations, then you treat the patient and he feels better and has fewer desaturations while he's waiting 3 months to go to a sleep laboratory, is there any harm in doing that? Do you have to do polysomnography? I know that 20% of the patients may have narcolepsy, but if the patient is better with the treatment, and you can institute it within 24 hours, it's certainly cost-effective.

Hill: Who is going to pay for it?

Bach: It costs practically nothing to just use oximetry . . .

Decker: You can't provide a CPAP machine to a patient until polysomnography's been done.

Stelmach: Medicare requires a PSG showing 30 apnea episodes or more during 6-7 hour sleep study.

Bach: Well, we haven't had a problem prescribing nasal ventilation without performing polysomnography in New Jersey.

Make: I suspect that's because you're not using it on Medicare pa-

tients, where you must have documentation.

Bach: No, Medicaid, Medicare, and private insurance pay for portable ventilators without polysomnography in New Jersey.

Make: I am not sure about Medicaid.

Mathews: I want to get back to the training issue; I fully sympathize with what you've been saying. About 2 years ago we instituted a polysomnography specialty for our senior baccalaureate respiratory care students. They don't become really good at these studies until almost a semester into the program when they're just about ready to graduate. It takes almost a semester, 4 months of doing this day in and day out, to get these students proficient, especially the critical thinking skills that they need to develop in order to do polysomnography and do it well.

Make: We've mentioned some potential conflict of interest here—not related so much to the respiratory care practitioner but more to the company or the vendor for which the respiratory care practitioner works. I think we mentioned sleep studies and whether it's at night or spot checks during the day. I'm wondering if there are comments about this because most of the companies that are doing the kind of monitoring at home are DMEs, and there is a potential conflict of interest. Do we have some suggestions or thoughts about that?

Sortor-Leger: I have one suggestion to avoid the conflict of interest, provide the monitoring through the hospital. I believe that the people who are doing the diagnosing and writing the prescriptions should be the people who provide the monitoring. I firmly believe that people who are benefiting financially from the equipment should have nothing to do with diagnosis and prescription. I knew Patrick (Dunne) would raise his hand on that one!

Dunne: Actually, just a comment and then a question to you, Mike (Decker)—first of all, I agree with your analysis that there's a lot of uncertainty in terms of who should be doing what and what capability they have. I think we need to understand that, as Sam (Giordano) said this morning, care follows cost. Now, the typical patient who presents with a sleep disorder, a classic obstructive sleep apnea, is not a Medicare patient. So, private insurance carriers are the ones who are going to be the big force in determining if we're going to have attended studies in the institution or in-home screening studies. I must tell you that there are several national insurance companies that shall remain nameless, who favor, strictly on a cost basis, unattended in-home monitoring, especially if the symptomatology of the patient leads to a very high probability that obstructive sleep apnea exists. I find that disconcerting. The other point, a question: Winding its way through the FDA right now is a new development known as smart-CPAP or demand-CPAP, or, I believe, D-PAP. Do you have any comments on where that might fit into the treatment regimen that has traditionally been used, either CPAP or BiPAP™?

Decker: We know about D-PAP, and we will face the same issues as we do with unattended monitoring. For instance, who should be setting this up in the home and instructing the patient? What variable is the D-PAP going to follow? Right now, it looks at changes in airflow; after the cessation of airflow and/or a drop in oxygen saturation, it increases its pressure automatically to try to restore airway patency. We still don't know if that helps with sleep fragmentation. If we treat hypoxia, and eliminate apnea, people feel better. So, is restoring oxygenation and airflow sufficient to treat the disease? It may be fine for some classic cases, but we just don't have enough information to generalize its use to all patients presenting with sleep complaints.

Kacmarek: I just wanted to make a comment on the issue Barry (Make) raised when we were discussing conflict of interest. I agree that there is a potential for conflict of interest with every home care company to whom you refer patients. I think the major problem, however, is that hospitals and practitioners referring patients to home care companies don't always take the time to carefully evaluate the company or take an active role in determining whether a particular company is appropriate, not only in the care that they provide but also whether they are capable of evaluating patients at home. We try to put a fair amount of time into screening to evaluate procedures and protocols of the companies that we deal with, and we do cross-checking. There's no reason why you can't compare what you see in the office to what's done in the home. I think you have to depend on the practitioner who is seeing the patient in the home to provide you feedback as to the patient's overall status. It's difficult to say when you do a home sleep study, who should evaluate it. It seems to me that you could have one company do the study, have an independent practitioner evaluate the results of that study, and make the decision on whether therapy is indicated, and then have the initial company go out and initiate the therapy. I believe there are ways of getting around this whole issue and eliminating or at least minimizing the potential for conflict of interest. We're all concerned about it, but I don't think it's an insurmountable issue. The same thing is true when you look at the training needed to set up a sleep study in the home. I have to believe that we're not expecting the average respiratory care practitioner who sets up

a home sleep evaluation to do the detailed analysis of the results. I have to believe that the data are going back to some qualified physician to interpret. The practitioner in the home is being taught how to set it up, how to monitor it, how to institute it appropriately, but it's not that individual who is evaluating the results. So it seems to me that, yes, it requires training, but I'm not sure that the average registered therapist who has experience cannot be reasonably well trained in a short period of time, as Nick (Hill) indicated, to perform this type of therapy in the home. I think we're exaggerating out of proportion what's necessary when you take a skilled practitioner and prepare that skilled practitioner to perform this study.

Celli: I have only a question for Michael (Decker) and Rick (Fletcher). We know who to treat when they're on the tip of the iceberg—the patient who's flagrant. My question to you is, What guideline should we use for the big bulk of borderline individuals whose sleep studies we review? You had trouble with your study in terms of showing a certain level of desaturation that ended up with a high mortality. Do we know the natural history of sleep apnea for the big group with moderate disease?

Fletcher: We don't have good prospective studies, but we will have in about 5 years from the Wisconsin group and also from Dr Susan Redline in Cleveland. We'll have a better handle on survival and outcome in untreated patients, which is very important. We have some retrospective, poorly controlled studies, but they're all we have to go by now. Basically, the cutoff from He[1] is 20 apneas per hour, and above that there's a higher mortality and lower survival in untreated patients. My problem is—along the lines of your question—it doesn't matter what we try to do with those patients who are in that gray area. It's what they want to do with therapy when you give it to them. Because it's becoming very apparent and pretty well demonstrated that they don't comply with nasal CPAP, and we don't have a good alternative. So, what's the point in going out and diagnosing the bottom of the iceberg when we don't have good treatment for them that they'll comply with?

REFERENCE

1. He J, Kryger MH, Zorick FJ, Conway W, Roth T. Mortality and apnea index in obstructive sleep apnea: experience in 385 male patients. Chest 1988; 94(1):9-14.

Hardy: I could bring up a whole set of issues related to pediatrics and sleep apnea, which also occurs in children. The whole differential diagnosis is different. The classic case is actually tonsillar and adenoidal hypertrophy. Classic treatment is T & A (tonsillectomy and adenoidectomy). All of these same issues challenge caregivers: Should they be studied in a sleep laboratory, can sleep labs accept kids, could they be studied at home? Not every kid needs a T & A. Decision making based just on a history of snoring is notoriously inaccurate. Adding a videotape of what the child looks like when they sleep is also inaccurate. Even ASDA labs—that can evaluate a child and try to figure out from the story, the physical exam, a videotape, before an actual polysomnogram—are wrong half the time. We often don't know who is really going to have physiologic compromise during sleep until we study them. Added to this whole muddle is the fact that in California, the insurer often demands a polysomnogram to act. People can't just get a T & A anymore. You can't do it based on a history and exam. You have to have this study. Additionally, other insurers may have fixed contracts with DME or home care companies who may not be qualified to assess young children.

Decker: In Cleveland we have a similar problem. We're a tertiary care center. We can make the diagnosis of sleep apnea with modified tests and bioparameter tests. The problem is getting CPAP. A lot of our patients are Medicare/Medicaid, and they will not be given a machine unless we have a polysomnography with EEG of at least 6 hours, documenting at least 30 apneas/hour. So regardless of how we make the diagnosis, we can't provide the therapy without that study.

Leger: Just one comment regarding D-PAP. I'm not sure that the D-PAP currently being looked at in the U.S. is the same device that we are studying in France, but if it is there are two interesting aspects with this device that I would like to elaborate on. The first point is that intelligent CPAP, as I know it, has the ability to continuously monitor the pressure needed to stop a patient's apneas and hypopneas and adjust the delivered flow accordingly throughout the sleep period. In addition, this device has a memory of each session of treatment and it is possible to retrieve this memory, which provides not only the exact history of the changes in flow and pressure but also the actual compliance of use. I think two very interesting points can be understood from this information; how does the patient's respiratory pattern change night-to-night and how does patient adherence with treatment influence the patient's symptoms. With this information, it may be possible to have a better approach to treatment. I think this evolution in treatment will be very interesting.

The American Respiratory Care Foundation Awards for 1994

Allen & Hanbury Literary Awards

$2,000 for the best original paper (study, evaluation, or case report) accepted for publication from December 1993 through October 1994. This award is not limited to papers based on OPEN FORUM presentations.

Four awards of $1,000 each for papers accepted for publication from November 1993 through October 1994 based on any OPEN FORUM presentation (not limited to 1993 OPEN FORUM).

Five awards of $500 each for the best papers submitted (not necessarily published) by 1994 OPEN FORUM participants who have 'never published' in the Journal. The never-published first author must present the abstract at the Annual Meeting and must submit a paper based on the abstract before the 1994 Annual Meeting (received in the Editorial Office by November 1, 1994). Co-authors may have previously published in RESPIRATORY CARE.

Dr Allen DeVilbiss Literary Award

$2,000 cash plus travel expenses to the AARC Annual Meeting to receive the award—for the best paper published from November 1993 through October 1994 that addresses new technology or a new application of current technology in respiratory care.

Radiometer America Literary Awards

Three awards of $333 each are to be awarded to the authors of the three best features from Test Your Radiologic Skill, Blood Gas Corner, Kittredge's Corner, and PFT Corner accepted for publication from November 1993 through October 1994. All three (or none) of the features may be chosen from a specific category (eg, all three may be chosen from Blood Gas Corner).

OPEN FORUM 1994

It's last call to submit your abstracts for possible presentation at the OPEN FORUM during the AARC Annual Meeting in Las Vegas, Nevada, December 10-13, 1994. The final deadline is May 28; abstracts must be postmarked by May 28 to be accepted for review. For more information on changes to the rules/instructions, see Page 405 of this issue.

Annual Meeting Registration Reimbursement

As in the past, any 1994 OPEN FORUM presenter (or co-author designee) will receive complimentary registration for an adequately prepared paper based on his 1994 OPEN FORUM abstract, submitted prior to or at the 1994 Annual Meeting. (If submitted on-site and accepted for review, convention registration will be reimbursed.)

All awards will be made at the 1994 Annual Meeting. Papers are judged automatically. No application is necessary.

THE NATIONAL BOARD FOR RESPIRATORY CARE—1994 Examination and Fee Schedule

	Examination Date	Application Deadline
CRTT:	July 16	May 1
	November 12	September 1
RRT:	June 4	February 1
	December 3	August 1
CPFT:	June 4	April 1
RPFT:	December 3	September 1

Fee Schedule	
Entry Level CRTT—new applicant:	$190.00
Entry Level CRTT—reapplicant:	$160.00
RRT Written and Clinical Simulation—new applicant:	$190.00
Written Registry Only—new applicant:	$ 90.00
Written Registry Only—reapplicant:	$ 60.00
Clinical Simulation Only—new & reapplicant	$100.00
Entry Level CPFT—new applicant:	$100.00
Entry Level CPFT—reapplicant:	$ 80.00
Advanced RPFT—new applicant:	$150.00
Advanced RPFT—reapplicant:	$130.00

	Active	Inactive
CRTT Recredentialing:	$25.00	$ 60.00
RRT Recredentialing:		
Written Registry Examination	$25.00	$ 60.00
Clinical Simulation Examination	$65.00	$100.00
CPFT Recredentialing:	$25.00	$ 80.00
RPFT Recredentialing:	$25.00	$130.00
P/P Specialty Credentialing:	$25.00	$130.00
Membership Renewal:		
CRTT/RRT/CPFT/RPFT		$ 12.00

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A VHA Satellite Network Production

Therapist-Driven Protocols: Implementation • March 29
Featuring James K. Stoller, MD
with Moderator Sam P. Giordano, MBA, RRT

Discusses therapist-driven protocol (TDP) implementation strategies, research, staff selection, training, obstacles, attitudes of other staff, and the results of TDP implementation.

New Strategies for Asthma Management • May 11
Featuring Randall C. Rosenblatt, MD
with Moderator David J. Pierson, MD

Presents patient self-management techniques and education, peak flow meter use, NAEP therapy goals, medication delivery devices, medications, and emergency room procedures.

Blood Gas and Related Measurements: Laboratory versus Bedside Devices • July 26
Featuring Barry R. Shapiro, MD
with Moderator Richard D. Branson, RRT

Presents the various means of obtaining blood gas information, invasive and noninvasive devices, the need for such devices, and when they should be used.

Hospital Operational Restructuring and Respiratory Care • September 13
Featuring Michael Boroch
with Moderator Sam P. Giordano, MBA, RRT

Reviews restructuring products, their implementation results, their relationship to respiratory care, and ways practitioners and managers can influence them.

The New Ventilator Management: Permissive Hypercapnia and other Variations on Conventional Mechanical Ventilation • November 8
Featuring Neil R. MacIntyre, MD
with Moderator Richard D. Branson, RRT

Outlines techniques and theories including pressure limited ventilation, reduced peak pressure, permissive hypercapnia, weaning and imposed work of breathing, and the next generation of ventilators.

Not-for-profit organizations are offered a free advertisement of up to eight lines to appear, on a space-available basis, in Calendar of Events in RESPIRATORY CARE. Ads for other meetings are priced at $5.50 per line and require an insertion order. Deadline is the 20th of the month two months preceding the month in which you wish the ad to run. Submit copy and insertion orders to Calendar of Events, RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229-4593.

Calendar of Events

AARC & AFFILIATES

May 3-6 in Grand Rapids, Michigan. The MSRC holds its 1994 Scientific Symposium at the Amway Grand Plaza Hotel. Contact MSRC, PO Box 950, East Lansing MI 48826-0950, or call Caroline Kimmel (517) 336-7605.

May 4-5 in Rockport, Maine. The MSRC presents the annual "Spring Fling" Seminar at the Samoset Resort. Topics include near drowning, mask CPAP, bronchodilator protocols, echocardiography, and medical ethics in critical care. Contact Peter Leavitt RRT RPFT, Respiratory Care Department, St Mary's Regional Medical Center, Campus Ave, Lewiston ME 04240. (207) 777-8538.

May 10-13 in Cincinnati, Ohio. Region II for Respiratory Care hosts its 21st Annual Conference at the Albert B Sabin Convention Center and Hyatt Hotel in Cincinnati, Ohio. Region II is sponsored by the Indiana, Ohio, and Kentucky Societies for Respiratory Care. This year's program, one of the largest respiratory care meetings in the nation, appeals to a wide variety of practitioners and managers with current topics and nationally recognized speakers. Program topics range from therapist-driven protocols to the care of the critically ill patient. Contact Priscilla Lethig (812) 426-8188.

May 11 AARC Videoconference. The AARC, in conjunction with VHA Satellite Network, presents the third of a 6-part videoconference series titled "Professor's Rounds in Respiratory Care." The third presentation is entitled "New Strategies for Asthma Management" (1 CRCE credit). For information call (214) 830-0061.

May 13-14 in Dallas, Texas. The American Association for Respiratory Care presents a special conference entitled "Therapist-Driven Protocols: The State-of-the-Art," a comprehensive review in three parts followed by a panel discussion; 12 hours of CRCE credit are awarded. Contact the AARC Convention Dept, 11030 Ables Lane, Dallas TX 75229-4593. Call (214) 243-2272, or fax (214) 484-2720.

May 14-17 in Tacoma, Washington. The 21st Annual Pacific Northwest Regional Respiratory Care Conference is held at the Sheraton Tacoma Hotel, Tacoma, Washington. Specialty section presentations cover topics in management, PFTs, pediatrics, and education. A golf tournament and sputum bowl are also held, and the latest in health care equipment is on display for 2 days. Contact Bob Bonner, Chairman of Health, Education and PE Division, Highline Community College, PO Box 98000, Des Moines WA. (206) 878-3710, ext 469.

May 31-June 3 in Myrtle Beach, South Carolina. The Georgia/South Carolina Region VI Committee presents its annual conference and assembly at the Landmark Hotel. Topics include Hospital Restructuring, Health Care Reform, Nitric Oxide, and Artificial Hemoglobin. Contact Lynda Thomas-Goodfellow at (404) 651-1498, or write to Region VI Committee, 275L Harbison Blvd, Suite #2, Columbia SC 29212.

May 31-June 3 in Orlando, Florida. The FSRC presents the annual Sunshine State Seminar in Orlando, Florida. This 4-day seminar provides topics from critical care to patient-focused care, with special exploration of topics related to HIV/AIDS. CEUs have been applied for, and a 4-day participant can receive 21 CEUs. Our social calendar includes, among other things, a golf tournament, and an evening at Sea World. Contact Marlene Thomas (904) 734-3320, ext 1290, or fax (904) 738-3102.

June 1-3 in Des Moines, Iowa. The ISRC annual conference is held at Hotel Savery in downtown Des Moines. Keynote speaker Senator Charles Grassley addresses health care reform. Contact Mike Wheeler (515) 263-5108.

June 8-10 in Checotah, Oklahoma. The OSRC presents its 29th Annual State Seminar and Exhibition at Fountainhead Resort and Convention Center on Lake Eufaula. This year's theme, "The Times–They Are A' Changing," addresses anticipated changes in the healthcare delivery system and critical issues for respiratory care. Other activities include a Manager's Mixer and the traditional golf tournament. For information on the seminar or exhibits, contact Karen Hart RRT at (405) 273-6442

June 15-17 in St Charles, Illinois. The ISRC presents its 26th annual state convention at Pheasant Run Resort. This year's theme is "ISRC Game Plan: Team Spirit '94" and features Sam Giordano MBA RRT, Executive Director of the AARC, as the keynote speaker. Mr Giordano speaks on "Health Care Reform and the RCP." New this year are breakfast sessions with choices of four topics: "CLIA Update," "OSHA Update," "Hospital-Based Nicotine Intervention Program," and "Reimbursement Issues for Home Medical Equipment Suppliers." The convention includes many other educational topics, an entry level review, and a resort setting for your enjoyment and relaxation. Contact Douglas McQueary (312) 962-4060 or Jane Reynolds (312) 883-6535.

July 26 AARC Videoconference. The AARC, in conjunction with VHA Satellite Network, presents the fourth of a 6-part videoconference series titled "Professor's

Rounds in Respiratory Care." The fourth presentation is entitled "Arterial Blood Gas Trouble Shooting: Interpretation, Analysis, and Quality Control" (1 CRCE Credit). For information call (214) 830-0061.

September 13 AARC Videoconference. The AARC, in conjunction with VHA Satellite Network, presents the fifth of a 6-part videoconference series titled "Professor's Rounds in Respiratory Care." The fifth presentation is entitled "Hospital Operational Restructuring and Respiratory Care" (1 CRCE Credit). For information call (214) 830-0061.

OTHER MEETINGS

May 6-7 in Philadelphia, Pennsylvania. The National Laboratory Training Network hosts a conference entitled "The Medical, Economic, and Regulatory Issues Affecting Patient-Focused Testing" at the Radisson Airport Hotel. The registration fee is $325, which includes lunch on both days and continuing education fees. Contact National Laboratory Training Network, PO Box 500, Eaton PA 19341-0500. (215) 263-8500.

May 10-11 in Little Rock, Arkansas. Respiratory Care Services of Arkansas Children's Hospital presents the 1994 Diamond Conference. Special sessions focus on neonatal/pediatric airway topics and pre-ECMO strategies in acute respiratory failure, including nitric oxide, HFOV, and conventional ventilation. The meeting also features a poster session, lunch tutorials, and roundtable discussions. Contact Mike Anders RRT, Respiratory Care Services, Arkansas Children's Hospital, 800 Marshall St, Little Rock AR 72202. (501) 320-3535, fax (501) 320-3411.

May 20-21 in Las Vegas. The 1994 Respiratory Health Conference takes place at Bally's Hotel & Casino. Topics are patient-focused care/therapist-driven protocols; health care reform; infectious diseases; and medical/legal ethics. Cost $130.00. Contact Bunny Grangaard at the American Lung Association of Nevada, PO Box 44137, Las Vegas NV 89116. (702) 454-2500, fax (702) 641-5466.

August 31-September 3 in Christchurch, New Zealand. The Third Congress of the Asia Pacific Association for Respiratory Care presents "Breathing into 2001," at the Christchurch Town Hall, Christchurch, New Zealand. Contact Stephanie K Humphries, The Planit Group Limited, Event Organizers, 201 Cambridge Terrace, Christchurch, New Zealand. (643) 366-5955, fax (643) 366-5944.

Neonatal Respiratory Care IISP

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1994 Call for Abstracts

RESPIRATORY CARE • OPEN FORUM

The American Association for Respiratory Care and its science journal, RESPIRATORY CARE, invite submission of brief abstracts related to any aspect of cardiorespiratory care. The abstracts will be reviewed, and selected authors will be invited to present papers at the OPEN FORUM during the AARC Annual Meeting in Las Vegas, Nevada, December 10-13, 1994. Accepted abstracts will be published in the November 1994 issue of RESPIRATORY CARE. Membership in the AARC is not necessary for participation.

SPECIFICATIONS—READ CAREFULLY!

An abstract may report (1) an **original study,** (2) the **evaluation of a method or device,** or (3) a **case or case series.** Topics may be aspects of adult acute care, continuing care/ rehabilitation, perinatology/pediatrics, cardiopulmonary technology, health occupations education, or management of personnel and health-care delivery. The abstract may have been presented previously at a local or regional—but not national—meeting and should not have been published previously in a national journal. The abstract will be the only evidence by which the reviewers can decide whether the author should be invited to present a paper at the OPEN FORUM. Therefore, *the abstract must provide all important data, findings, and conclusions.* Give specific information. Do not write such general statements as "Results will be presented" or "Significance will be discussed."

Essential Content Elements

An **original study** abstract *must* include (1) Introduction: statement of research problem, question, or hypothesis; (2) Method: description of research design and conduct in sufficient detail to permit judgment of validity; (3) Results: statement of research findings with quantitative data and statistical analysis; (4) Conclusions: interpretation of the meaning of the results. A **method/device evaluation** abstract *must* include (1) Introduction: identification of the method or device and its intended function; (2) Method: description of the evaluation in sufficient detail to permit judgment of its objectivity and validity; (3) Results: findings of the evaluation; (4) Experience: summary of the author's practical experience *or* a notation of lack of experience; (5) Conclusions: interpretation of the evaluation and experience. Cost comparisons should be included where possible and appropriate. A **case report** abstract *must* report a case that is uncommon or of exceptional teaching/learning value and must include: (1) patient data case summary and (2) significance of case. Content should reflect results of literature review. The author(s) should have been actively involved in the case and a case-managing physician must be a co-author or must approve the report.

Abstract Format and Typing Instructions

Accepted abstracts will be photographed. First line of abstract should be the title in all capital letters. Title should explain content. Follow title with names of all authors (including credentials), institution(s), and location. Underline presenter's name. Type or electronically print the abstract *single spaced in the space provided on the abstract blank.* Insert only one letter space between sentences. Text submission on diskette is encouraged but must be accompanied by a hard copy. Identifiers will be masked (blinded) for review. Make the abstract all one paragraph. Data may be submitted in table form *and simple figures may be included provided they fit within the space allotted. No* figures, illustrations, or tables are to be attached to the abstract form. Provide all author information requested in right column of abstract form. A clear photocopy of the abstract form may be used. Standard abbreviations may be employed without explanation. A new or infrequently used abbreviation should be preceded by the spelled-out term the first time it is used. Any recurring phrase or expression may be abbreviated if it is first explained. Check the abstract for (1) errors in spelling, grammar, facts, and figures; (2) clarity of language; (3) conformance to these specifications. An abstract not prepared as requested may not be reviewed. Questions about abstract preparation may be telephoned to the editorial staff of RESPIRATORY CARE at (214) 243-2272.

Deadlines

Deadline Allowing Revision

Authors may choose to submit abstracts early. Abstracts received by March 15 will be reviewed and the authors notified by April 22. Rejected abstracts will be accompanied by a written critique that should in many cases enable authors to revise their abstracts and resubmit them by the final deadline (May 28).

Final Deadline

The mandatory Final Deadline is May 28 (postmark). Authors will be notified of acceptance or rejection by *letter only*— to be mailed by August 15.

Mailing Instructions

Mail (Do not fax!) 2 clear copies of the completed abstract form and a stamped, self-addressed postcard (for notice of receipt) to:

RESPIRATORY CARE OPEN FORUM
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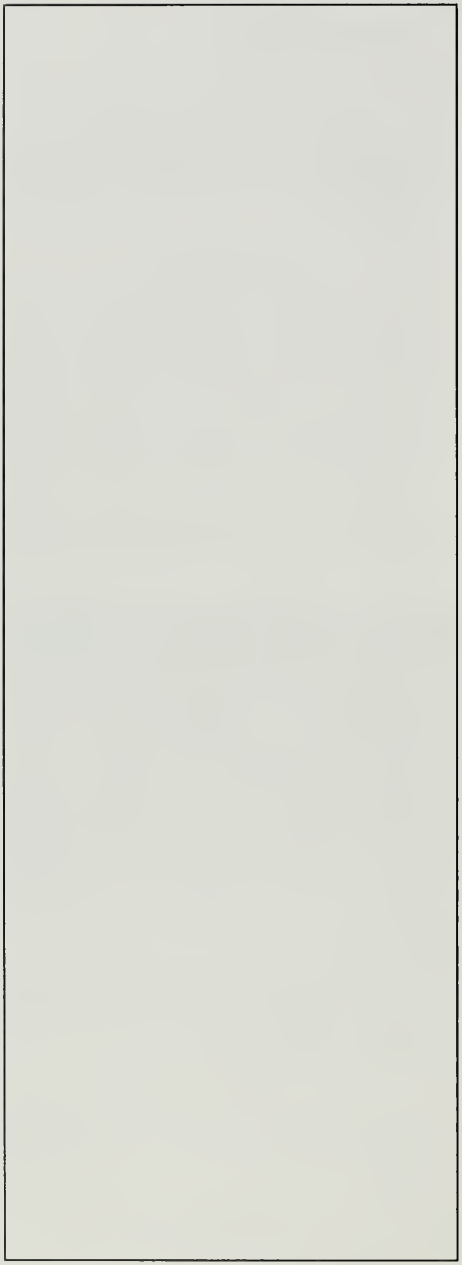
1994 Respiratory Care Open Forum

Abstract Form



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2. Follow title with *all* authors' names including credentials (underline presenter's name), institution, and location.
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Early deadline is March 15, 1994
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Final deadline is May 28, 1994
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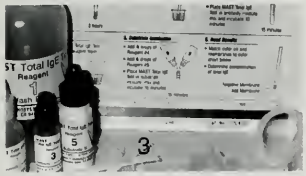

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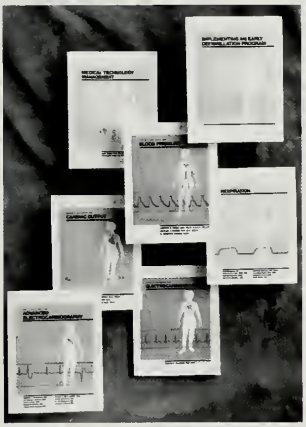
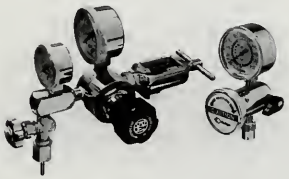
CYLINDER CARRIER. WT Farley Inc introduces the new 20 cylinder carrier. This mobile cylinder carrier is designed to move cylinders safely; and the best thing about it is (according to the manufacturer)—once you have one, you will never have to worry about safely transporting cylinders again; it is easily used by all healthcare workers, regardless of strength. The cart has a unique 6-wheel design that makes it easily maneuverable around corners and in and out of elevators; it can rotate 360° within its own space. The cart's dimensions are 42″×28″×25″ and it is constructed of epoxy coated steel. The model ES120W Mobilox 20 sells for $250. For more information write WT Farley Inc, Dept RC 4450 Shopping Lane, Simi Valley CA 93063, (800) 327-5397. Please mention RESPIRATORY CARE when you call.



BIOPHYSICAL MEASUREMENT BOOKS. The newest addition to the SpaceLabs Medical collection of biophysical measurement books is "Medical Technology Management." According to the company, the books provide biomedical professionals and clinicians with information about specific physiologic parameters and monitoring systems, thus allowing them to optimize their use and interpretation. Designed for easy reference, each book is illustrated with dozens of drawings and includes detailed bibliographies, glossaries, and references. The company currently offers six other books on topics such as blood pressure, cardiac output, electrocardiography, respiration, implementing an early defibrillation program, and advanced electrocardiography. For information and prices, call Karyn Beckley (206) 882-3700 and mention RESPIRATORY CARE when you call.



INSTRUMENT-QUALITY REGULATORS. The OEM Custom Medical Regulator Division of Western Enterprises announces two new OEM regulator series. Series OEM 3000 presents instrument-quality regulators used to calibrate blood-gas analysis equipment. Features include single-stage operation with nonmetallic diaphragm flow controls for accurate and immediate calibration readings; dual CC/M, BAR, and LPM gauge scales for world-wide application; balanced pressure seat/nozzle design with ± 0.3 LPM precision; durable Kel-F seat; double inlet filtering; and an extended, limited warranty. Series 444CXX are newly designed suction regulators that feature a dual-scale, high visibility vacuum gauge face, stainless steel diaphragm for high-precision applications, premium chrome plating for superior appearance, and easy repairability due to fewer working parts. For detailed information, write Western Enterprises, OEM Custom Medical Regulator Division, Dept RC, 875 Bassett Road, Westlake OH 44115-1196, (216) 871-2160. Please mention RESPIRATORY CARE when you call.

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Please complete the card below

AARC Membership No. _____

Old Address

Name _____

Street _____

City/State/Zip _____

New Address

Street _____

City/State/Zip _____

Check the boxes below for information from the AARC

- ❑ Change of address
- ❑ AARC Membership Info
- ❑ AARC Catalog
- ❑ AARC Position Statement

RESPIRATORY CARE

April Reader Service **Expires July 31, 1994**

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Institution _____

Street _____

City/State/Zip _____

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80	81	82	83	84	85	86	87	88	89	90	91	92	93	94
95	96	97	98	99	100	101	102	103	104	105	106	107	108	109
110	111	112	113	114	115	116	117	118	119	120	121	122	123	124
125	126	127	128	129	130	131	132	133	134	135	136	137	138	139
140	141	142	143	144	145	146	147	148	149	150	151	152	153	154
155	156	157	158	159	160	161	162	163	164	165	166	167	168	169
170	171	172	173	174	175	176	177	178	179	180	181	182	183	184
185	186	187	188	189	190	191	192	193	194	195	196	197	198	199

I. Type of Instn/Practice
1 ❑ Hosp ≥ 500 or more beds
2 ❑ Hosp 300 to 499 beds
3 ❑ Hosp 200 to 299 beds
4 ❑ Hosp 100 to 199 beds
5 ❑ Hosp <100 or less bed
6 ❑ Skilled Nursing Facility
7 ❑ Home Care Practice
8 ❑ School
II. Department
A ❑ Respiratory Therapy
B ❑ Cardiopulmonary
C ❑ Anesthesia Service
D ❑ Emergency Dept
III. Specialty
1 ❑ Clinical Practice
2 ❑ Perinatal Pediatrics
3 ❑ Critical Care
4 ❑ Clinical Research
5 ❑ Pulmonary Function Lab
6 ❑ Home Care/Rehab
7 ❑ Education
8 ❑ Management
IV. Position
A ❑ Dept Head
B ❑ Chief Therapist
C ❑ Supervisor
D ❑ Staff Technician
E ❑ Staff Therapist
F ❑ Educator
G ❑ Medical Director
H ❑ Anesthesiologist
I ❑ Pulmonologist
J ❑ Other MD
K ❑ Nurse
V. Are you a member of the AARC?
1 ❑ Yes 2 ❑ No

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140	141	142	143	144	145	146	147	148	149	150	151	152	153	154
155	156	157	158	159	160	161	162	163	164	165	166	167	168	169
170	171	172	173	174	175	176	177	178	179	180	181	182	183	184
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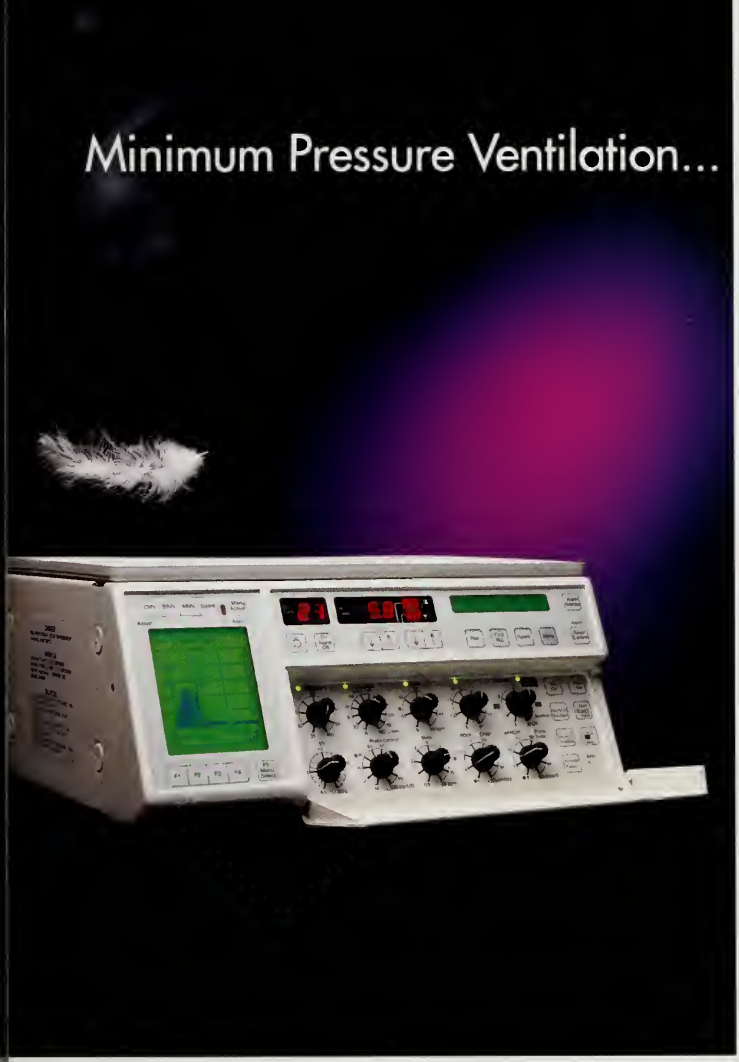

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